# ANTICARDIOLIPIN ANTIBODIES IN UNEXPLAINED PREGNANCY LOSS

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# M.D. BRANCH-II
# OBSTETRICS AND GYNAECOLOGY


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# CERTIFICATE

This is to certify that this dissertation titled **"ANTICARDIOLIPIN ANTIBODIES IN UNEXPLAINED PREGNANCY LOSS''** is a bonafide record of work done by **Dr.S.B. Melita Keren Glady** during the period of her Post graduate study from May 2006 to March 2009 under guidance and supervision in the Department of Obstetrics and Gynaecology, Raja Sir Savalai Ramasamy Mudaliar Hospital, Stanley Medical College Hospital, Chennai-600013 in partial fulfilment of the requirement for **M.D. Branch-II Obstetrics and Gynaecology** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in March 2009.

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# CONTENTS

# INTRODUCTION

# INTRODUCTION

Man has amazing interest in research and his thirst for knowledge has begun right from the era of Stone Age. But what perhaps escapes his mind is that the human body is the most fascinating thing available and demands more exploration than anything else.

The science of immunology is one such breath taking field and the ability of human being to discriminate between self and non-self antigens is phenomenal. The recognition and the response to foreign antigens, an ability perhaps most highly developed in mammalian species is one of the most intensively studied topics in science and medicine today.

Pregnancy is an unique immunological stage where a natural homeostasis exists between antigenically different tissues.

One of the Medawars initial hypothesis, on the maintenance of the foetal allograft was that pregnancy was associated with suppression of the

maternal immune response thereby allowing foetal survival. The pregnant host however must also maintain immunocompetence against both pathogenic and neoplastic invasion during the gestation to ensure host survival.

Autoimmunity is one deviation from this normal pattern, where antibodies are directed against one's own antigens. One of the fundamental questions in the study of any autoimmune disease concerns the role of autoantibodies in the pathogenesis of the disorder. In certain autoimmune conditions like Grave's disease and Myasthenia gravis the evidence that they are directly involved in the pathogenesis is strong.

Much recently interest has been focussed over the Antiphospholipid Antibody syndrome. Antiphospholipids were first linked to pregnancy loss more than 30 years ago and the condition known as antiphospholipid syndrome is perhaps the most convincing immune disturbance other than antierythrocyte and antiplatelet alloimmune disorders. Specific criteria for antiphospholipid syndrome have been delineated and the anticardiolipin assay has been standardized and authorities have agreed on a laboratory criteria to define lupus anticoagulant.

In the last 25 years, numerous investigators have described the association between antibodies that bind phospholipid molecules and repetitive pregnancy losses. Antiphospholipid antibodies account for 3% to 5% of patients with repetitive pregnancy losses. The frequency of fetal death and recurrent abortion in untreated patients with antiphospholipid antibodies is greater than 90%.[13]

The prevalence of antiphospholipid antibodies in general population is around 2-4%. Of patients with the antiphospholipid antibody syndrome, over 50% of them have the primary antiphospholipid antibody syndrome. In persons with systemic lupus erythematosus, around 30% will develop the antiphospholipid antibody syndrome. The two best characterised antiphospholipid antibodies, lupus anticoagulant and anticardiolipin antibodies have been widely recognised as risk factors for pregnancy related complications including recurrent pregnancy loss, intrauterine growth retardation, pre-eclampsia, antepartum haemorrhage and the failure of in-vitro fertilisation and embryo transfer, thus emerging as a clinical entity of "reproductive autoimmune failure syndrome".[2] In general, anticardiolipin

antibodies are more common than the lupus anticoagulant in patients with the antiphospholipid antibody syndrome.[6]

Nonetheless considerable confusion exists regarding antiphospholipid syndrome and related reproductive problems. The state of affairs primarily derives from two problems; the first is the premature introduction of non standardized antiphospholipid assays into clinical use, without proper standardization, as a result of which less well versed clinicians sometimes make the diagnosis of antiphospholipid syndrome in women who are negative for lupus anti coagulant and anticardiolipin antibody.

A second major problem is that of unwarranted discrepancies in the clinical and laboratory features of patients considered to have a diagnosis of antiphospholipid syndrome. This problem is most apparent in the case selection for screening and management protocol.

Our entire work revolves around,

a. Whether these anticardiolipin antibodies are a direct cause of pregnancy loss (or) merely an epiphenomenon.
b. Criteria for screening a woman for anticardiolipin antibodies

c. The kind of obstetric outcome and pregnancy loss that is common in patients who are anticardiolipin antibody positive.

Anticardiolipin antibody is found to have a higher predictive power than lupus anticoagulant for foetal loss (Pattison et al). According to Lynch et al and various other researchers an elevated IgG anticardiolipin antibody was the only antiphospholipid antibody to be significantly associated with the foetal demise. So IgG anticardiolipin antibody has been exclusively studied in this work.

# AIM OF STUDY

# AIM OF STUDY

To evaluate the presence of Anticardiolipin antibodies in women with unexplained pregnancy loss.

To assess the type of common obstetric outcome in Anticardiolipin Antibody positive women.

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

The incidence of habitual abortion is in the range of 0.4-0.8% and in approximately half of these cases a specific etiologic factor can be found. 3-5% of habitual abortion cases are thought to be due to autoimmune abnormalities. Anticardiolipin antibodies and lupus anticoagulant factor are the two antiphospholipid antibodies which have a role in this situation[4].

Cardiolipins are negatively charged phospholipids which are obtained from beef heart with alcoholic extraction. This is the basis of the flocculation test, VDRL, which has been used in the diagnosis of syphilis until recent years. The tissue damage in syphilis results in formation of some auto antibodies which react with cardiolipin and some other phospholipids. Today Lupus Anticoagulant Factor and AntiCardiolipin Antibody are found in many disorders characterized by arterial and venous

thrombosis, habitual abortion, intrauterine growth retardation and intrauterine fetal death[4].

Most sensitive tests for the detection of anticardiolipin antibodies are the solid phase RIA and ELISA techniques in which microplates covered with cardiolipin antigens react with anticardiolipin antibodies. Lupus Anticoagulant Factor is detected indirectly by prolongation of activated partial thromboplastin time test (APTT). It can also be detected by several other coagulation tests (Kaolin clotting time, Tissue thromboplastin inhibition test).

# HISTORY

Historically, antiphospholipid antibodies were first noted in patients who had positive tests for syphilis without signs of infection. Subsequently, a clotting disorder was associated with two patients with systemic lupus erythematosus in 1952. In 1957, a link between recurrent pregnancy loss and what is now called the lupus anticoagulant was established by Laurell and Nillson[6.] They described a patient with five prior intrauterine deaths who had a biologically false-positive syphilis test and an anticoagulant antibody. Later it was found that the circulating anticoagulant and the molecule

responsible for the false positive serology were antiphospholipid antibodies[13]. Ultimately, the lupus anticoagulant was further described in 1963 and in 1972 the term lupus anticoagulant was given. In 1983, Dr. Graham Hughes described the association between antiphospholipid antibodies and arterial as well as venous thrombosis.[6]

Feinstein and Rapaport (1972) introduced the term lupus anticoagulant (LAC) in a review of acquired inhibitors of coagulation. This was based on early recognition that certain patients with lupus had some coagulation tests that were prolonged and thus suggested anticoagulant activity. Paradoxically, the so called anticoagulant is powerfully thrombotic in vivo, although the LAC prolongs all phospholipid dependent coagulation tests, including the prothrombin time, partial thromboplastin time, Russell viper venom time, Each of these tests requires a phospholipid surface to which other clotting factors attach and combine. Thus detection of LAC is based indirectly on the prolongation of in vitro tests by this circulating antiphospholipid antibody. ACA is detected serologically using enzyme linked immunosorbent assay (ELISA). Most often IgM anticardiolipin antibodies that are found alone are stimulated by infections or drugs and are innocuous. (Silver et al)[14.]

Antiphospholipid Syndrome may be either primary if other features of connective tissue disease are absent or secondary to established connective tissue diseases such as systematic lupus erythematosus. It is argued that primary APS is misnomer, i.e., primary APS is only a Prodromal State before the patient develops other features of Autoimmune Disease – Secondary APS. More recent studies suggest that antibodies that really matter are beta2 glyco protein, the cofactor by which anticardiolipin binds to phospholipids. Patients who have cardiolipin antibodies in high titre usually have high levels of lupus anticoagulant.(Michael de Swiet et al). [11]

Cardiolipin antibodies may belong to both IgG and IgM subtypes. The IgG antibodies seem to be a better predictor of fetal outcome, although the presence of IgM antibodies is not without risk to the fetus. [11]

## *PREVALENCE OF ANTIPHOSPHOLIPID ANTIBODY*

Studies have reported that the prevalence of anticardiolipin antibody in women with three or more miscarriages is between 11% and 42% (BIRDSALL et al., BARBUE et al., CREAGH et al., PARKE et al., UNANDER et al.,) in contrast to prevalence of 2% in women with a low risk obstetric history.

| Category | No of Subjects | Lupus anticoagulant | Anticardiolipin Antibody | Reference |
|---|---|---|---|---|
| Low Risk Population | 933 | 1.00% | 1.20% | PATTISON et al.,[31] |
| Recurrent Miscarriage | 500 | 9.10% | 5.50% | RAI et al.,[32] |

In RAI et al.'s study IgG anticardiolipin antibody was found in 3.3% and prevalence of the IgM anticardiolipin was 2.2 %

EROGLU et al., in their research suggested that the prevalence of anticardiolipin antibody is very low in first trimester losses and is not very significant. [33] MACLEAN and colleagues in their study proved that anticardiolipin antibody had a prevalence of 8.2% in first trimester losses. [34] Bocconi from catholic university, Italy suggested that anticardiolipin antibody is found in 20.5% in those with unexplained pregnancy loss. [35] Nevertheless, APL antibodies are found in upto 5% of apparently healthy controls and upto 35% of patients with SLE.

## *PATHOPHYSIOLOGY OF ANTIPHOSPHOLIPID ANTIBODIES IN PREGNANCY[14]*

According to Charmley 1999, platelets may be damaged directly by antiphospholipid antibodies or indirectly by binding Beta 2 Glycoprotein I which causes platelets to be susceptible for aggregation. Rand et al 1997, proposed that phospholipids containing endothelial cell or syncytiotrophoblast membranes may be damaged directly by the antiphospholipid antibody or indirectly by antibody binding to either Beta 2 GlycoProtien1 or Annexin V. This prevents the cell membrane from protecting the syncytiotrophoblast and endothelial cells and results in exposure of basement membrane.

Pierro and Co-workers 1999 reported that antiphospholipid antibodies decrease the decidual production of the vasodilating prostaglandin $E_2$. Decreased Protein C or S activity as well as increased prothrombin activation has reported by Ogunyemi et al 2002. Recent studies suggest that uncontrolled placental complement activation by antiphospholipid antibodies may play a role in fetal loss and growth restriction.

# MECHANISM OF ACTION

The Placenta is severely infarcted and the foetus is often growth restricted. Even in the pregnancy of those foetuses that survived, severe preeclampsia is very common. Current theories centre round the damage to placental vascular endothelium caused by cardiolipin antibodies, platelet deposition and imbalance to thromboxane/prostacyclin production tilted towards too much thromboxane and too little prostacyclin all leading to inadequate maternal placental perfusion. [11]

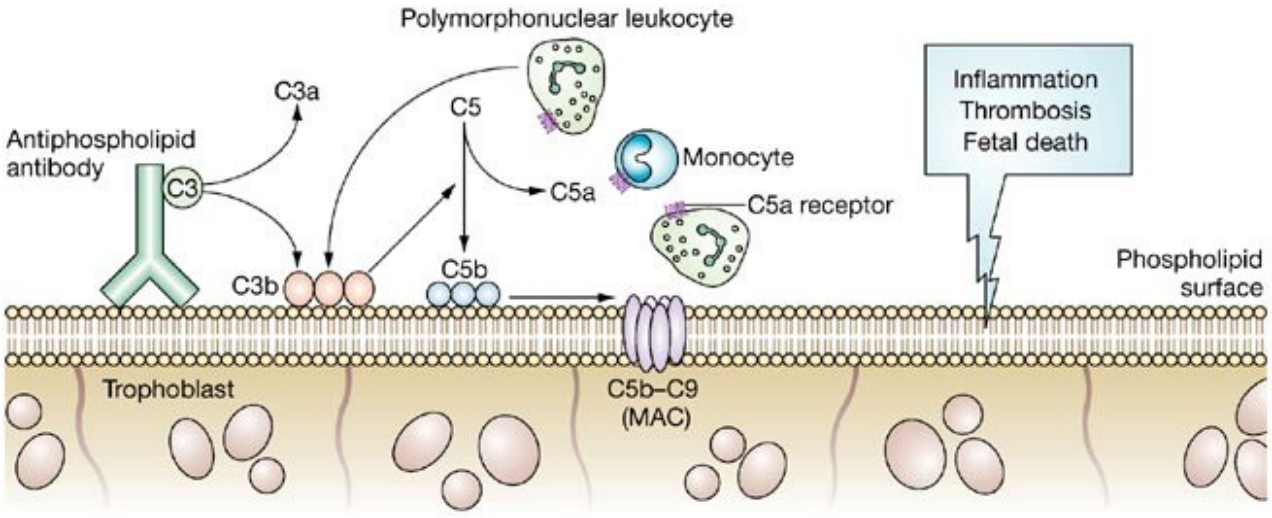
Inhibition of protein C and tissue plasminogen activator has also been postulated as mechanisms whereby lupus anticoagulant may cause thrombosis.

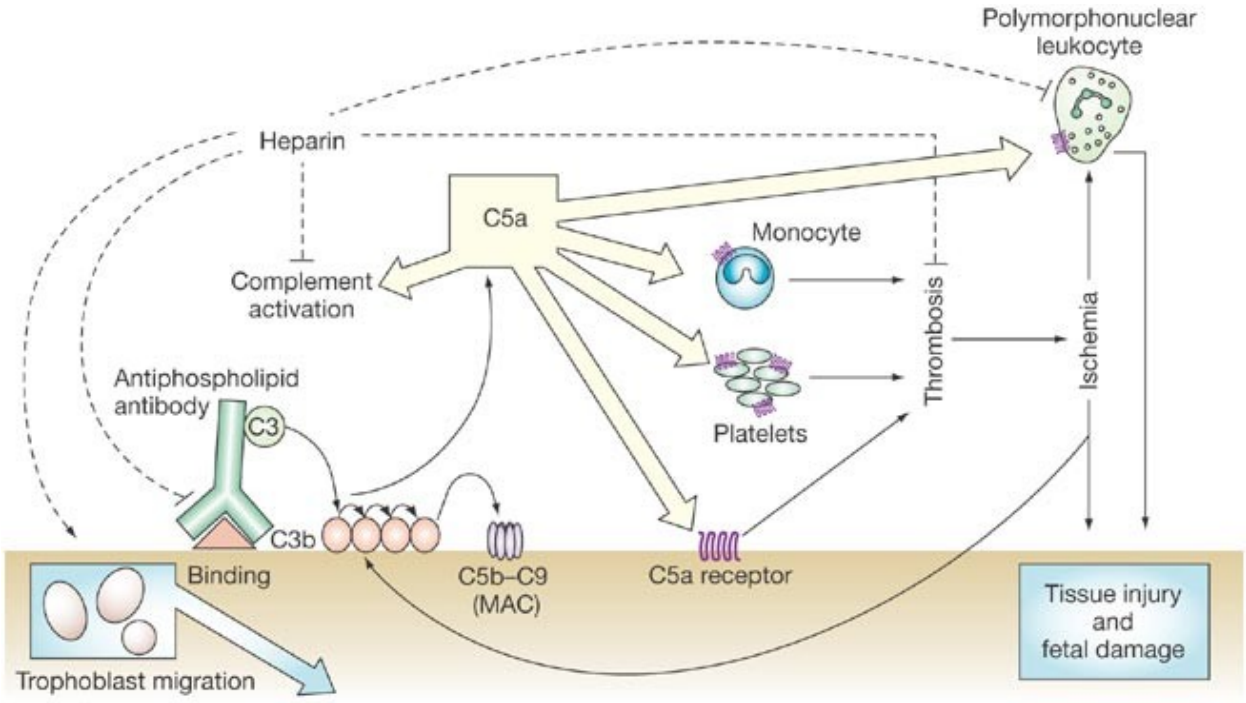
## MECHANISMS OF REDUCTION OF ANNEXIN V LEVELS AND ACCELERATION OF COAGULATION ASSOCIATED WITH ANTIPHOSPHOLIPID ANTIBODIES [14]

A relative lack of Annexin V a naturally occurring anti coagulant on the intervillous surfaces of the placenta may be a contributing factor.

Anionic phospholipids on the surface of cell membrane bilayer serve as potent co factors for assembly of three coagulation complexes—tissue factor-VIIa complex, IXa-VIIIa complex and Xa-Va complex. When antiphospholipid antibodies are absent, annexin V forms clusters that bind to the surfaces of anionic phospholipids and inhibit coagulation.

In antiphospholipid antibody syndrome, directly or through an interaction with protein phospholipid co-factors, they disrupt the ability of annexin V to cluster on the phospholipid surface. This reduces binding affinity of annexin V, which permits more anionic phospholipids to be available to form complexes with coagulation proteins. Coagulation is thus accelerated and thrombosis promoted.






# *DIAGNOSTIC CRITERIA OF ANTIPHOSPHOLIPID SYNDROME*[12]

The first classification criteria for the diagnosis of APS was formulated in conference workshop in October 1998 at Sapparo, Japan and referred to as Sapparo Criteria.

They have been recently revised, the major change being essentially the addition of anti beta 2 glyco protein 1 antibody as one of the laboratory criteria and documentation of persistence of the antibodies for a longer duration by repeating the test at 12 Weeks instead of 6 Weeks.

## *REVISED CLASSIFICATION CRITERIA FOR ANTIPHOSPHOLIPID SYNDROME*

**Clinical Criteria**

1. Vascular thrombosis: one or more episode of arterial, venous or small vessel thrombosis in any organ.

2. Pregnancy morbidity:
    a. One or more unexplained death of a morphologically normal foetus ≥ 10 Weeks of Gestation.
    b. One or more premature birth before 34 weeks because of eclampsia, preeclampsia or placental insufficiency.
    c. Three or more unexplained spontaneous consecutive abortion before 10 weeks of gestation with maternal anatomic, hormonal and paternal karyotype abnormalities excluded.

**Laboratory Criteria**

1. Lupus anticoagulant present in serum/plasma on two or more occasion atleast 12 weeks apart detected.
2. Anticardiolipin antibody of IgG and/or IgM isotype in serum or plasma present in medium or high titre on two or more occasions atleast 12 weeks apart.
3. Anti beta 2 Glyco protein 1 antibody of IgG and/or IgM isotype in serum or plasma present in two or more occasions atleast 12 weeks apart.

# *CLINICAL FEATURES OF THE ANTIPHOSPHOLIPID SYNDROME*[11]

**Thrombosis**

Venous

Recurrent deep vein thrombosis (also intravascular coagulation and retinal vein thrombosis)

**Arterial**

Cerebrovascular accidents

Peripheral arterial gangrene

Coronary thrombosis

Retinal artery thrombosis

**Other**

Pulmonary hypertension

avascular necrosis

**Abortion**

Recurrent IUD, placental thrombosis and infarction.

**Thrombocytopenia**

Intermittent, often acute

**Other occasional features**

Coombs positivity

Livido reticularis

Migraine

Chorea

Epilepsy

Chronic leg ulcers

Endocardial disease

Progressive dementia due to repeated cerebrovascular thrombosis

## *WHAT PROBLEMS DO APLA CAUSE?*[8]

**PREGNANCY COMPLICATIONS:**

APLA are present in approximately 10% to 20% of women with recurrent miscarriage. Women with APLA have a higher risk of pregnancy loss at or after 10 weeks of pregnancy. This is in contrast to pregnancy loss in the general population, which occurs more commonly before 10 weeks of pregnancy. Additionally APLA is associated with other

pregnancy complications, including eclampsia, preeclampsia, and placental insufficiency.

**VENOUS CLOTS:**

One of the complications of APLA are clots in the veins, most commonly either a Deep Vein Thrombosis in the leg or a pulmonary emboli in the lung. Less common locations for venous clots include the superficial veins (superficial thrombophlebitis), the eye, the abdomen, in or around the brain, and in or around the liver.

**ARTERIAL CLOTS:**

APLA may also cause clots in the arteries such as stroke, heart attack, and clots in the arteries of the arm, leg, eye, kidney or abdomen. Suspicion for the presence of APLA in a person with an arterial clot is higher if the person has no obvious risk factor for arterial disease (such as diabetes, high blood pressure, high cholesterol), does not smoke or is relatively young.

**OTHER CLINICAL PRESENTATIONS**

Many studies show that APLA can be associated with a variety of other clinical findings, including low platelets, anaemia, heart valve disease, skin rashes, mini-strokes, joint pain, joint inflammation, dry eyes and dry mouth.

## CATASTROPHIC APLA SYNDROME[8]

Most patients with APLA who develop clots will develop them as individual events and possibly have recurrent clots at a later time. However, a very small group of people with APLA develop multiple clots in different organ systems throughout the body within a matter of days. This is called catastrophic APLA syndrome. Clots may occur at the same time in the kidney, brain, heart, extremities, lungs and /or other organs, with resultant multi organ failure and high risk of mortality. The treatments are blood thinners, steroids, plasma exchange, and possibly suppression of the immune system.

## THE ASYMPTOMATIC INDIVIDUAL WITH APLA[8]

Some people with APLA will never develop blood clots or experience pregnancy loss. Few datas are available on the risk of blood clot

or pregnancy loss in an asymptomatic individual with APLA. One percent of people may develop a blood clot every year. There are presently no recommendations that support the routine use of blood thinners in asymptomatic people. However, one aspirin per day appears to be beneficial.

Asymptomatic individuals should take the following precautions:

a) Inform health care providers that they have APLA

b) Consider use of short term blood thinners to prevent a clot during situations those increase the risk of developing a clot (such as surgery or immobilization)

c) Know the symptoms of DVT ( swelling and pain throughout the leg, l warmth, and discoloration) and pulmonary embolism (sudden chest pain and shortness of breath)

d) Seek medical care immediately if symptoms of Deep vein thrombosis or Pulmonary edema develop and inform the physicians the presence of APLA in their blood.

e) Modify their risk factors for arterial and venous blood clots, including avoiding hormone therapy, not smoking, normalizing weight, and controlling blood pressure, cholesterol and blood sugar.

# *RISKS OF ANTIPHOSPHOLIPID SYNDROME AND PREGNANCY*

## THROMBOTIC COMPLICATIONS OF ANTIPHOSPHOLIPID SYNDROME IN PREGNANCY[4]

Numerous retrospective studies confirm a link between Antiphospholipid syndrome and venous or arterial thrombosis. Approximately 70% of thrombotic events occur in the venous system, although arterial thrombosis and cerebrovascular accidents are also common. Transient CNS manifestations of ischemia are also common in APLA patients. Antiphospholipid antibodies are present in approximately

2% of individuals with unexplained thrombosis and are the only identifiable pre disposing factor in 4% to 28% of cases of stroke in otherwise healthy patients younger than age 50. The life time risk of thrombo embolism in women with APLA is unknown. However over half occur in relation to pregnancy or in use of combination oral contraceptives.

Central Nervous system involvement is one of the most prominent clinical manifestations and includes arterial and venous

thrombotic events, psychiatric features, and other non thrombotic neurological syndromes (Sanna et al 2003). Interestingly, APLA may mimic the presentation of multiple sclerosis (Ruiz-Irastona et al, 2001). [14]

## OBSTETRIC COMPLICATIONS OF ANTIPHOSHPHOLIPID SYNDROME IN PREGNANCY[4]

### 1. GESTATIONAL HYPERTENSION/ PREECLAMPSIA

The median rate of gestational hypertension/ preeclampsia in pregnancies complicated by APLA is 32%, with a range upto 50%. Preeclampsia may develop as early as 15 to 17 weeks gestation. The rate of gestational hyper tension/ preeclampsia does not appear to be markedly diminished by treatment with either glucocorticoids and low dose aspirin or heparin and low dose aspirin. Two groups of investigators have reported that women with early onset, severe preeclampsia are more likely to test positive for aPL antibodies compare to healthy controls. Based on these findings testing for aPL antibodies should only be considered in early onset, severe preeclampsia.

### 2. UTERO PLACENTAL INSUFFICIENCY AND PRETERM BIRTH

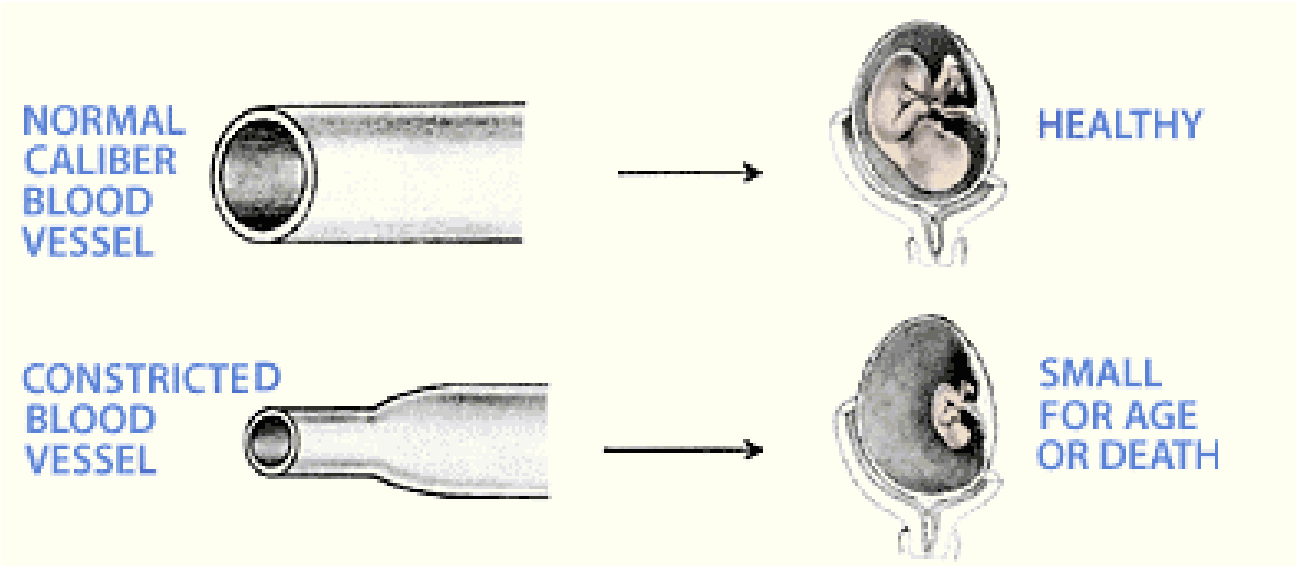
Several investigators have found relatively high rates of IUGR in association with aPL antibodies. Even with currently used treatment protocols, the rate of IUGR approaches 30%. Pregnancies complicated by APS are also most likely to exhibit non reassuring fetal heart rate patterns during antenatal tests of fetal well being and intra partum monitoring. Not surprisingly, the rate of pre term birth in these series ranges from 32% to 65%.

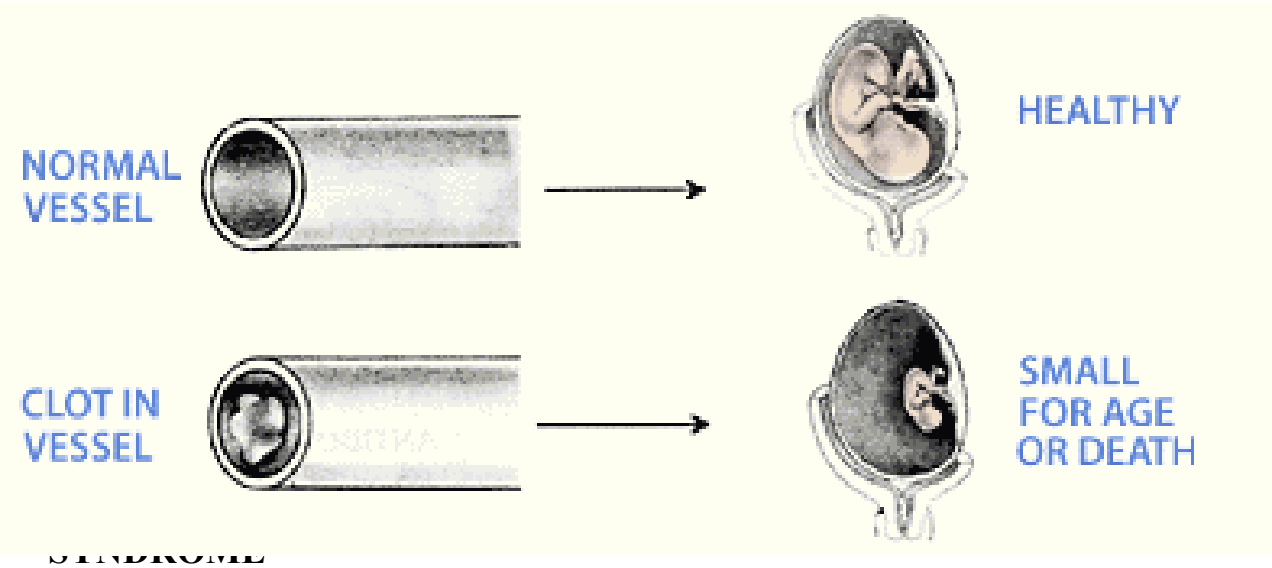
## 3. PREGNANCY LOSS[4]

Atleast 40% of pregnancy loss reported by women with lupus anticoagulant or medium to high positive IgG anticardiolipin antibodies occur in the fetal period. According to recent prospective studies, the rates of obstetric complications were relatively low, with fetal death, preeclampsia, and pre term birth occurring in 4.5%, 10.5% and 10.5% respectively. Only 1 of 300 women suffered a thrombotic event, and no neonatal deaths due to complications of prematurity were reported.

Fetal demise can occur at any stage of pregnancy and there is an association with severe preeclampsia, intrauterine growth retardation and placental abruption. The reported fetal loss rate is of the order of 80% for those women with raised titres of anti cardiolipin antibodies. [5]

According to Oshiro et al 1996 and Roque et al 2001 fetal deaths are more common than first trimester miscarriages. Polzin et al (1991) identified antiphospholipid antibodies in a fourth of 37 women with growth restricted foetuses. Branch et al (1989) found a 16% incidence of






SYNDROME

Catastrophic APLA is rare but devastating syndrome characterised by multiple simultaneous vascular occlusions throughout the body, often resulting in death. The diagnosis should be suspected if atleast 3 organ

systems are affected and confirmed if there is histopathologic evidence of acute thrombotic microangiopathy affecting small vessels. Renal involvement occurs in 78% of patients.

Most have hypertension; 25% eventually require dialysis. Other common manifestations described by Asherson include adult respiratory distress syndrome (66%) cerebral microthrombi and microinfarct(56%), myocardial microthrombi(50%), dermatological abnormalities (50%) and disseminated intravascular coagulation (25%). Death from multi organ failure occurs in 50% of patients. Although no treatment has shown in to be superior to another, a combination of anticoagulants (usually heparin)and steroids plus either plasmapheresis or IVIG has been successful in some patients. Streptokinase and urokinase have also been used to treat acute vascular thrombosis.

## Indications to test for Antiphospholipid antibodies[14]

1. Recurrent pregnancy loss
2. Unexplained second or third trimester loss
3. Early onset severe Preeclampsia
4. Venous or Arterial thrombosis

5. Unexplained fetal growth restriction
6. Autoimmune or Connective tissue diseases
7. False positive serological test for syphilis
8. Prolonged coagulation studies
9. Positive auto antibody test

## LABORATORY TESTS LIKELY TO BE ABNORMAL IN PATIENTS WITH ANTIPHOSPHOLIPID SYNDROME[51]

- Partial thromboplastin time (PTT).
- PTT remains prolonged when the patient's plasma is mixed with normal plasma. Positive "mixing study"
- Kaolin clotting time (KCT)
- Russell viper venom time (RVVT)
- Low platelet count
- Anticardiolipin antibody IgG and IgM

- Beta 2 glycoprotein I dependent Anticardiolipin antibody
- Serologic tests for syphilis
- Antinuclear antibody

## WHO SHOULD BE TESTED FOR APLA AND HOW OFTEN? [8]

People who have had one of the clinical events listed in the diagnostic criteria and who do not have other identifiable reasons for blood clots or pregnancy losses should be tested for APLA. If they have found to have APLA, the test should be rechecked atleast 12 weeks later. If the second test is again positive, they can be categorised as having APLA syndrome. If a person has one positive APLA test followed by a negative APLA test 12 weeks later, the test should be repeated again in 6 months. If the test is negative twice, the person does not have APLA syndrome.[8]

## HOW DO YOU TEST FOR APLA?

There are two ways to test the blood for the presence of APLA. One test is a direct measurement of the amount of antibody present. Because the immune system can make APLA against a variety of different phospholipids

or proteins bound to phospholipids (for example, cardiolipin, beta2 glycoprotein I), a variety of tests can be performed. Furthermore, because our immune system can make three different forms of antibodies, referred to as immunoglobulin (IgG, IgM, IgA), three different tests can be performed for each phospholipids. These tests are poorly standardized, which cause problems when interpreting the results or comparing results from one laboratory to those of another.[8]

The other way to look for the presence of APLA is by measuring the effect that the antibodies have on the clotting system in the test tube (lupus anticoagulant test). A variety of clotting tests are used for this purpose, most commonly the Rusell viper venom (RVVT)-based test and the Lupus anticoagulant partial thromboplastin time (LA-PTT) based test.[8]

Although anticardiolipin antibodies are more common than lupus anticoagulants, the presence of a lupus anticoagulant puts a person at high risk of having a clot than does the presence of anticardiolipin antibodies alone. For a person who has anticardiolipin antibodies, the higher the antibody level, the greater the risk of developing a blood clot.[8]

# *MANAGEMENT OF ANTIPHOSPHOLIPID SYNDROME DURING PREGNANCY*[4]

The ideal treatment for APLA during pregnancy should include:

1. Improvement in maternal and fetal – neonatal outcome by preventing Pregnancy loss, pre eclampsia, placental insufficiency and preterm birth

2. Reduction or elimination of the risk of thromboembolism.

At present, maternally administered heparin is considered as treatment of choice. It is usually initiated in the early first trimester after ultrasonographic demonstration of a live embryo. In most case series and trials, low dose aspirin is included in the treatment regimen.

## SUBCUTANEOUS HEPARIN REGIMENS USED FOR ANTIPHOSPHOLIPID SYNDROME DURING PREGNANCY[4]

## 1. PROPHYLACTIC REGIMENS

Recommended in women with no history of thrombotic events-diagnosis of recurrent preembryonic and embryonic loss or prior fetal death

or early delivery because of severe preeclampsia or severe placental insufficiency.

**STANDARD HEPARIN**

7500-10000 U q12hourly in the first trimester, 10000U q 12hourly in the second and third trimester.

**LOW MOLECULAR WEIGHT HEPARIN**

1. Enoxaprin 40 mg once daily or dalteparin 5000U once daily.
2. Enoxaprin 30 mg q 12 h or dalteparin 5000 U q 12h

## 2. ANTICOAGULATION REGIMEN

Recommended in women with a history of thrombotic events.

**STANDARD HEPARIN**

Every 8-12 hours, adjusted to maintain the midinterval heparin levels in the therapeutic range.

**LOW MOLECULAR WEIGHT HEPARIN**

1. Weight adjusted (enoxaparin 1mg/kg q 12 or dalteparin 200U/kg q 12h )
2. Intermediate dose (enoxaparin 40 mg once daily or dalteparin 5000U once daily until 16 weeks of gestation and q 12h from 16 weeks on).

Many centres are now using low molecular weight heparin, since it has increased bio availability and a longer half life and can therefore conveniently be given once daily. It seems to have no greater deleterious effect on bone density than occurs physiologically during pregnancy.[10]

Women with APLA should be counselled about the potential risks of heparin therapy during pregnancy, including heparin induced osteoporosis and heparin induced thrombocytopenia(HIT).

Osteoporosis resulting in fracture occurs in 1% to 2% of women treated during pregnancy. Women treated with heparin should be

encouraged to take daily supplemental calcium and vitamin D (Eg. Prenatal vitamins). It also seems prudent to encourage daily axial skeleton weight bearing exercise (Eg. Walking). LMWH is much less likely to be associated with Heparin induced thrombocytopenia, compared with unfractionated sodium heparin.

IVIG has also been used in pregnancy complicated by APLA, especially women with particularly poor past histories or recurrent pregnancy loss during heparin treatment.[4] Immunoglobulin is administered intravenously in doses of 0.4 g/kg daily for 5 days. This is repeated monthly or it is given as a single dose of 1g/kg each month. The drug may cause anaphylactic reactions, especially in women who have IgA deficiency due to anti IgA antibodies[14].

Hydroxychloroquine has been shown to diminish the thrombogenic properties of APL antibodies in a murine thrombus model.

Healthy women with recurrent embryonic and preembryonic loss and low titres of APLA don't require treatment. The controlled trial of Pattison et al included majority of such women and found no difference in live birth rate in using low dose aspirin or placebo.

## PRE-PREGNANCY

Women with APLA should seek pre conception counselling. Patients should be informed about the risks of heparin induced osteoporosis. And recommendations for appropriate protective measures should be provided. Primary APLA patients should be assessed for anaemia, thrombocytopenia and renal disease.

## PRENATAL

An early transvaginal ultrasound is useful to confirm an intrauterine pregnancy and for accurate dating. One of the anticoagulation prophylaxis should be started. Calcium supplementation should be encouraged. Prenatal

visits should occur every 2 to 4 weeks until 20 to 24 weeks gestation and every 1 to 2 weeks thereafter. Because of the risk of IUGR and oligohydramnios, serial ultrasound examination should be performed every 3 to 4 weeks after 17 to 18 weeks gestation. Antenatal surveillance should be initiated at 30 to 32 weeks.

## LABOUR AND DELIVERY

Patients receiving prophylactic anticoagulation with heparin can be instructed to withhold their injections at the onset of labour. Alternatively injections can be discontinued 12 hrs before a planned induction. In cases of extremely high risk for thromboembolism intravenous heparin can be started in labour and discontinued 2 to 4 hrs prior to anticipated delivery. Intravenous heparin can be resumed 4 to 6 hrs after vaginal delivery and 12 hrs after caesarean.

## POSTPARTUM

Warfarin thromboprophylaxis should be initiated as soon as delivery with doses adjusted to achieve an international normalised ratio of 3. Both heparin and warfarin are safe for breast feeding mothers. Finally oral contraceptives containing oestrogens are absolutely contraindicated

## RESULTS OF TREATMENT

Although improved outcomes are reported with some of the treatments, Branch et al (2003) cautioned that recurrent fetal loss is still 20 to 30%. In one study, Lockshin et al reported 23 of 32 women with prior fetal death and antiphospholipid antibody greater than 40Ig G units had a recurrent fetal death despite treatment with prednisone, aspirin or both. Current data suggest that most efficacious therapy to be low dose heparin-7500 to 10,000 units administered subcutaneously, twice daily given along with low dose aspirin, 60 to 80 mg once daily. If active lupus also is present, then prednisone is usually also given usually.[14]

Retrospective case review of 82 consecutive pregnancies in women with antiphospholipid syndrome with varying treatment combination, including prednisolone, aspirin, heparin and immunoglobulin infusion. Outcomes were similar in all treatment groups, with 73% live birth, 60% Intra uterine growth retardation, 50% preeclampsia or fetal distress, 37% delivering preterm and 5% post natal thromboembolism. Number of previous pregnancy failures was the only significant predictor of adverse pregnancy outcome ($p=0.02$). [50]

# MATERIALS AND METHODS

# MATERIALS AND METHODS

This study on anticardiolipin antibodies in unexplained pregnancy loss was carried out in 200 women with previous pregnancy loss in Govt. RSRM Hospital. Study period was 2006-2008. The 200 women were of random gestational age.

The selection criteria were taken as follows:

i) Recurrent miscarriage ≥3

ii) Second or third trimester pregnancy loss which includes all intrauterine demises.

iii) Placental abruption

iv) H/O early onset preeclampsia

v) Intrauterine growth retardation

vi) SLE

vii) Thrombocytopenia

Exclusion criteria were as follows:

i) Anemia

ii) Diabetes mellitus

iii) Chronic Hypertension

iv) Renal Disorder

v) Sexually transmitted disorder

vi) Rh incompatibility

vii) Uterine anomalies

viii) Fetal anomalies

ix) Syphilis

About 5ml of serum was collected from the patients in a plain test tube.

Anticardiolipin antibodies encompass IgG, IgM and IgA class of antibodies, but in this study semi quantitative and qualitative determination of IgG antibodies in serum was done.

## *PRINCIPLE OF A PROCEDURE*

An indirect Non-competitive enzyme immunoassay for the semi quantitative and qualitative determination of cardiolipin IgG antibodies is performed. The wells of the microtitre plate are coated with cardiolipin antigen. Antibodies specific for cardiolipin present in the patient's sample bind to the antigen.

The second step, the antigen antibody complex reacts with an enzyme labelled second antibody (Enzyme Conjugate) which leads to the formation

of an enzyme labelled antigen antibody sandwich complex. The enzyme label converts added substrate from a colored solution.

The rate of colour formation from the chromogen is a function of enzyme conjugate complexed with the bound antibody and this is proportional to the initial concentration of the respective antibodies in the patient's sample.

## Materials Required

| | |
|---|---|
| Standards | 6 vials of cardiolipin antibody standards at concentrations of 0-4-8-20-50-100 GPL- u/ml |
| Positive Control | 1 vial |
| Negative Control | 1 vial |
| Wash buffer | |
| Sample buffer | |
| Conjugate | 1 vial of antihuman IgG horse radish per-oxidase conjugate |
| Enzyme Substrate | 1 vial of Tetra methyl benzene |
| Microtitre well strips | |
| Microtitre plate reader | |
| Distilled $H_2O$ | |

| | |
|---|---|
| Glass ware | |
| Micro pipettes (10, 50, 100, 1000µl)<br><br>or multi pipette. | |

# Procedure

1. Wash wells one time with wash buffer immediately prior to use.
2. Dilute Serum/plasma (1:101)
3. Dispense 100µl of standards, controls and diluted patient samples in to appropriate wells.
4. Incubate for 30 minutes
5. Aspirate fluid from wells and wash wells 3-5 times with wash buffer.
6. Dispense 100µl of conjugates in to all wells.
7. Incubate for 30 minutes.
8. Aspirate fluid from wells and wash wells 3-5 times with wash buffer
9. Dispense 100µl of Enzyme substrate in to all wells.
10. Incubate for 10 minutes in the dark.
11. Dispense 50µl of stop solution in to all wells.
12. Read absorbance (OD) at 450 nm within 30 minutes of adding the stop solution

# *INTERPRETATION OF RESULTS*

Photometric quantitation is made at 450 nm using a microtitre plate photometer. A standard curve is obtained from the linear plot of normalized optical densities (OD Mean/O.D max 1) (i.e.) OD of the individual standard samples divided by the O.D. of the highest standard. The concentrations of the control and patient samples are then determined by comparing the normalized optical densities for each unknown to the standard airne and reading of the corresponding concentrations.

For the assessment of patient's sera the following range is recommended.

Negative : <12 GPL u/ml

Equivocal : 12-18 GPL u/ml

Positive : >18 GPL u/ml

# OBSERVATION

# OBSERVATION

This study regarding Anti-Cardiolipin antibodies in unexplained pregnancy loss was carried out on 200 pregnant women with previous pregnancy wastage. They were selected after ruling out the other possible causes such as Diabetes, syphilis, etc. Detection of these anti-cardiolipin antibodies was done by measuring the IgG antibodies by Elisa method. Values of more than 18 GPL u/ml were taken as positive.

**TABLE 1**
**AGE DISTRIBUTION OF SUBJECTS**

| Age in Years | No. of Patients | Percentage |
|---|---|---|
| 18-20 | 30 | 15% |
| 21-25 | 92 | 46% |
| 26-30 | 52 | 26% |
| 31-35 | 26 | 13% |

Table I shows age distribution of 200 patients with unexplained pregnancy loss.

Maximum number of patients belong to the age group 21-25 [46%] followed by the age group 26-30[26%]. 15% belong to age group 18-20 and 13% along to age group 31-35.

## AGE DISTRIBUTION OF SUBJECTS

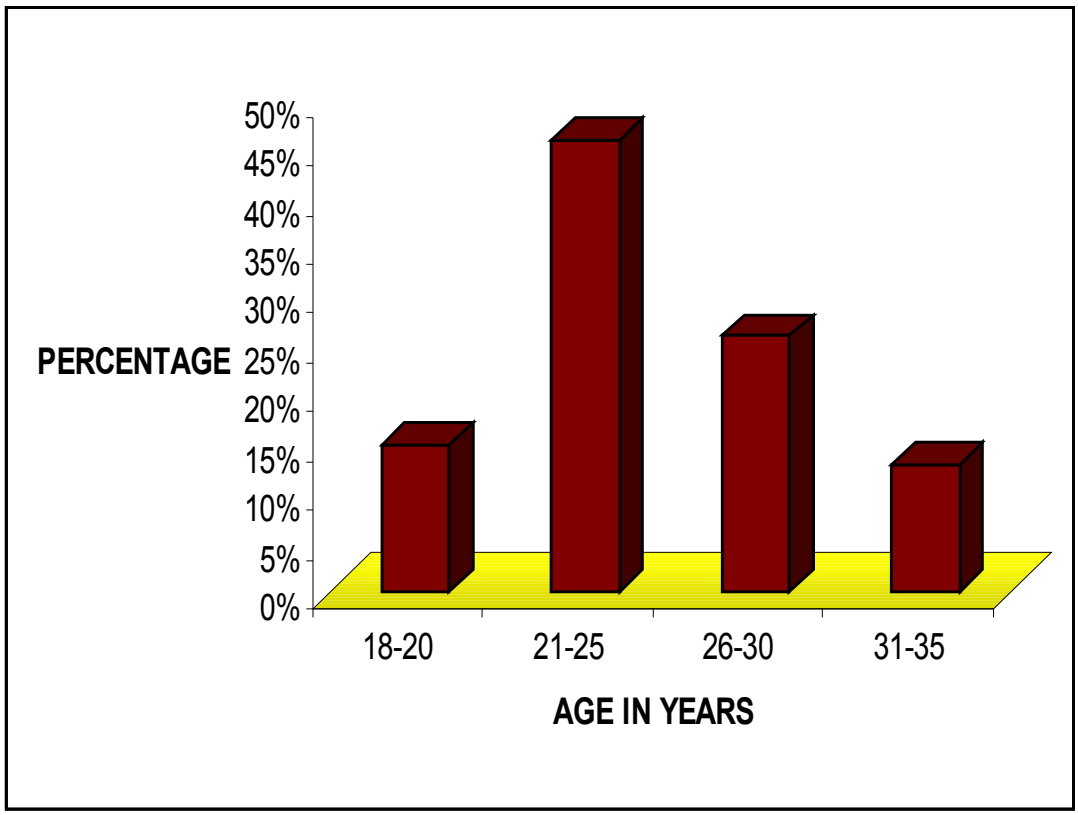


**TABLE II**

**CONCEPTION DISTRIBUTION OF SUBJECTS**

| Gravida | No. of Subjects | Percentage |
|---|---|---|
| PRIMI Gravida | 8 | 4% |
| II | 78 | 39% |
| III | 62 | 31% |
| IV | 36 | 18% |
| V | 12 | 6% |
| VI & above | 4 | 2% |

Table II shows the distribution of conceptions among the test subjects. Maximum no. of cases were under the group of Gravida II [39%]. This was followed by Gravida III group which was about 31%. 18% were IV gravida, 6% were V gravidas 4% were primis and 2% VI gravida and above.

# CONCEPTION DISTRIBUTION OF SUBJECTS

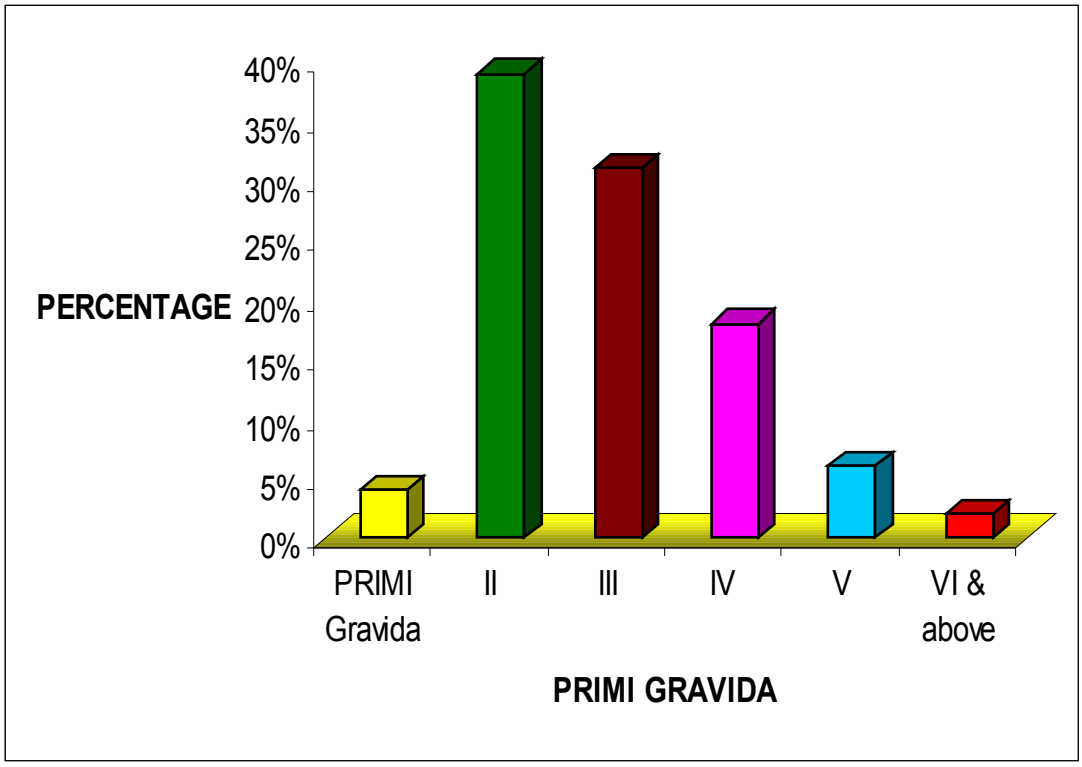


**TABLE III**

**GESTATIONAL AGE DISTRIBUTION OF SUBJECTS**

| Gestational Age | No.of Subjects | Percentage |
|---|---|---|
| I Trimester | 20 | 10% |
| II Trimester | 22 | 11% |
| III Trimester | 158 | 79% |

Table III shows dramatic accumulation under III trimester which tallies around 79%. 11% of cases were in the II trimester and 10% of cases in the I trimester.

# GESTATIONAL AGE DISTRIBUTION OF SUBJECTS

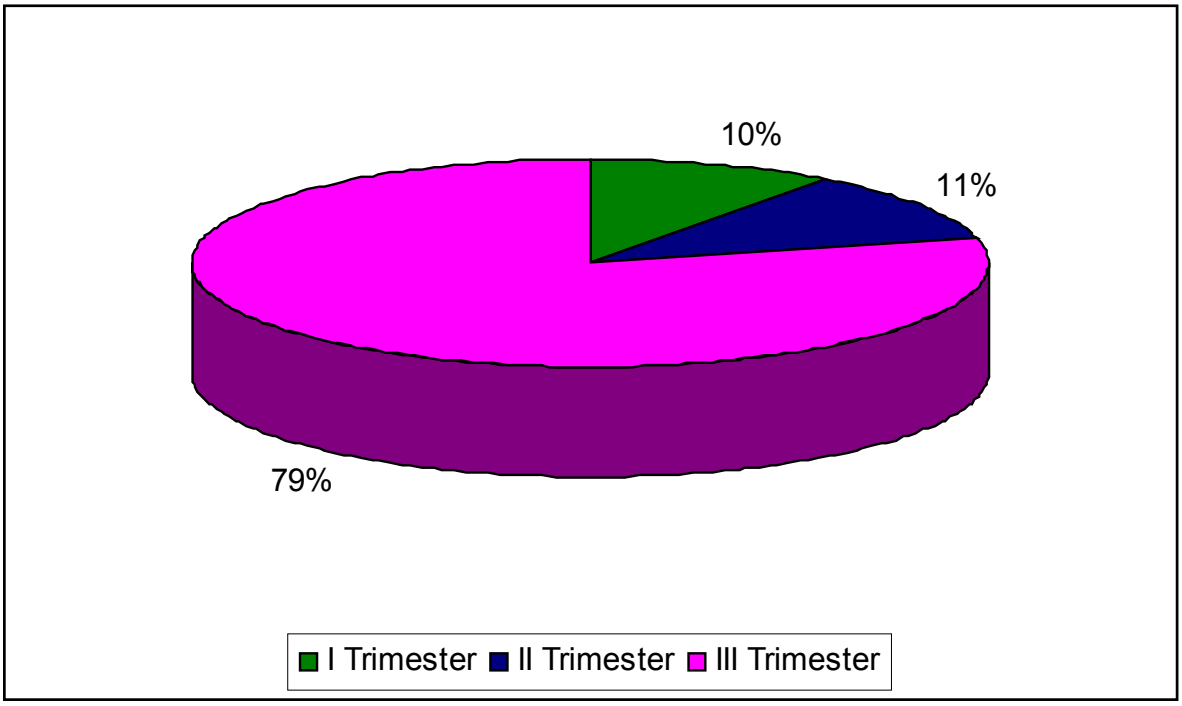

10%
11%
79%
I Trimester
II Trimester
III Trimester

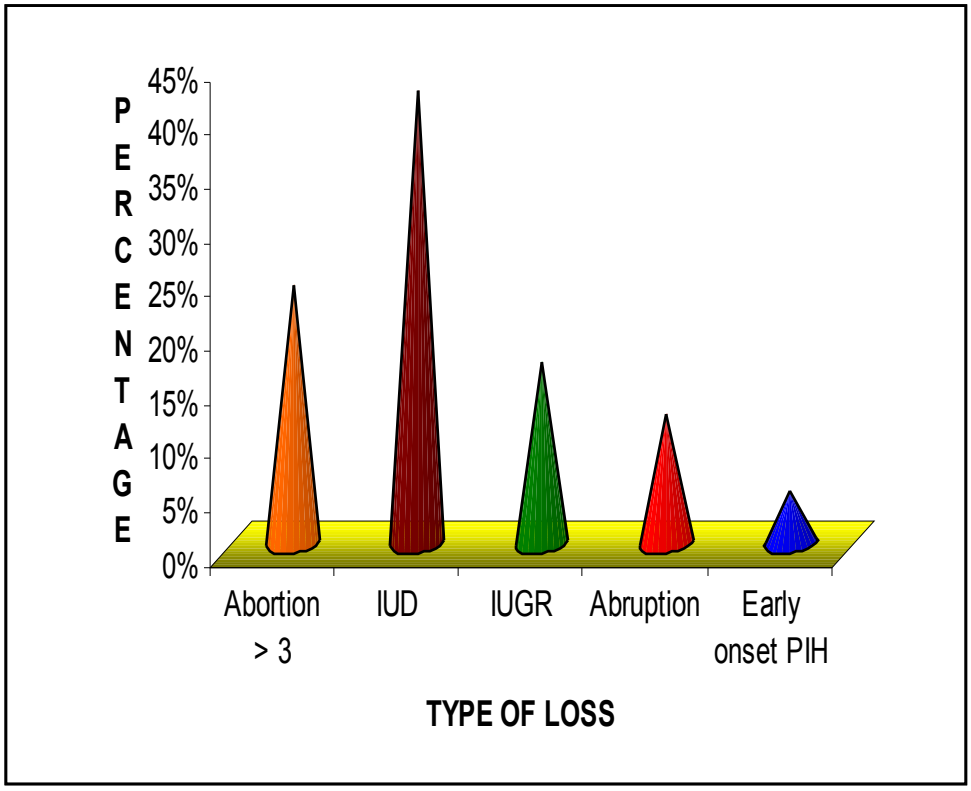
**TABLE IV**

**OBSTETRIC HISTORY OF SUBJECTS**

| Type of Loss | No. of Cases | Percentage |
|---|---|---|
| Abortion ≥ 3 | 48 | 24% |
| Intrauterine demise | 84 | 42% |
| Intrauterine growth retardation | 34 | 17% |
| Abruption | 24 | 12% |
| Early onset PIH | 10 | 5% |

Table IV shows the obstetric history of subjects taken for the study. Maximum numbers of cases included were intrauterine demise 42% followed by abortion ≥ 3 24%, intrauterine growth retardation 17%, abruption 12%, early onset PIH 5%.

OBSTETRIC HISTORY OF SUBJECTS


PERCENTAGE
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
Abortion > 3
IUD
IUGR
Abruption
Early onset PIH
TYPE OF LOSS

NOW CONSIDERING THE POSITIVE CASES WHICH TURNED OUT TO BE EIGHTEEN OF TWO HUNDRED.

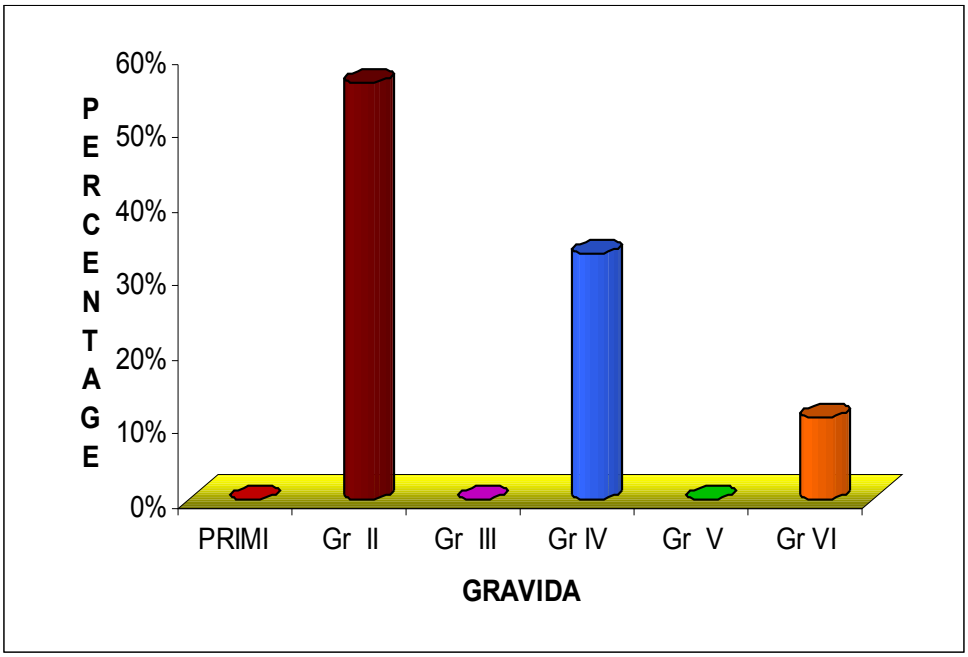
**TABLE V**

**CONCEPTION DISTRIBUTION OF POSITIVE CASES**

| Gravida | No. of Subjects | Percentage |
|---|---|---|
| PRIMI Gravida | - | - |
| Gr II | 10 | 56% |
| Gr III | - | - |
| Gr IV | 6 | 33% |
| Gr V | - | - |
| Gr VI | 2 | 11% |

55.5% of the positive cases happened to be 2nd gravida followed by 4th gravida who occupied 33%. 11.1% were gravida VI and none in the primi or gravida V

## CONCEPTION DISTRIBUTION OF POSITIVE CASES


PERCENTAGE
60%
50%
40%
30%
20%
10%
0%
PRIMI
Gr II
Gr III
Gr IV
Gr V
Gr VI
GRAVIDA

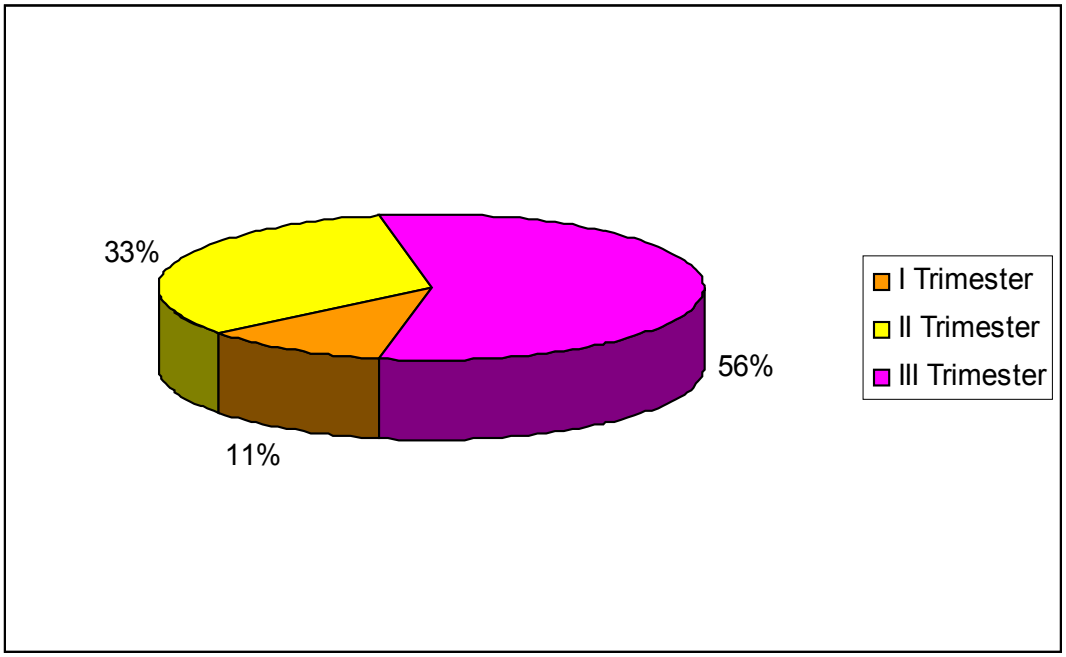
## TABLE VI

## GESTATIONAL AGE DISTRIBUTION IN POSITIVE CASES

| Gestational Age | No.of Subjects | Percentage |
|---|---|---|
| I Trimester | 2 | 11% |
| II Trimester | 6 | 33% |
| III Trimester | 10 | 56% |

The above important table shows that maximum number of cases presented to us at the third trimester [55.5%] followed by 33.3% in II trimester and 11.1% in I trimester.

## GESTATIONAL AGE DISTRIBUTION IN POSITIVE CASES




## TABLE VII

## OBSTETRIC HISTORY IN POSITIVE CASES

| Type of Loss | No. of Cases | Percentage |
|---|---|---|
| Abortion ≥ 3 | 2 | 11% |
| Intrauterine demise | 8 | 45% |
| Intrauterine growth retardation | 2 | 11% |
| Abruption | 2 | 11% |
| Early onset PIH | 2 | 11% |
| Mixed Presentation | 2 | 11% |

This table VII gives an insight in to the obstetric history in the positive cases. The majority were intrauterine deaths (44.4%) and abortions. Intrauterine growth retardation babies, Abruptio placenta and early onset preeclampsia all taking on equal share of 11.1%. Mixed presentation (i.e.) abruption and preelcampsia or abruption and live births etc. also occupied 11.1%

## OBSTETRIC HISTORY IN POSITIVE CASES

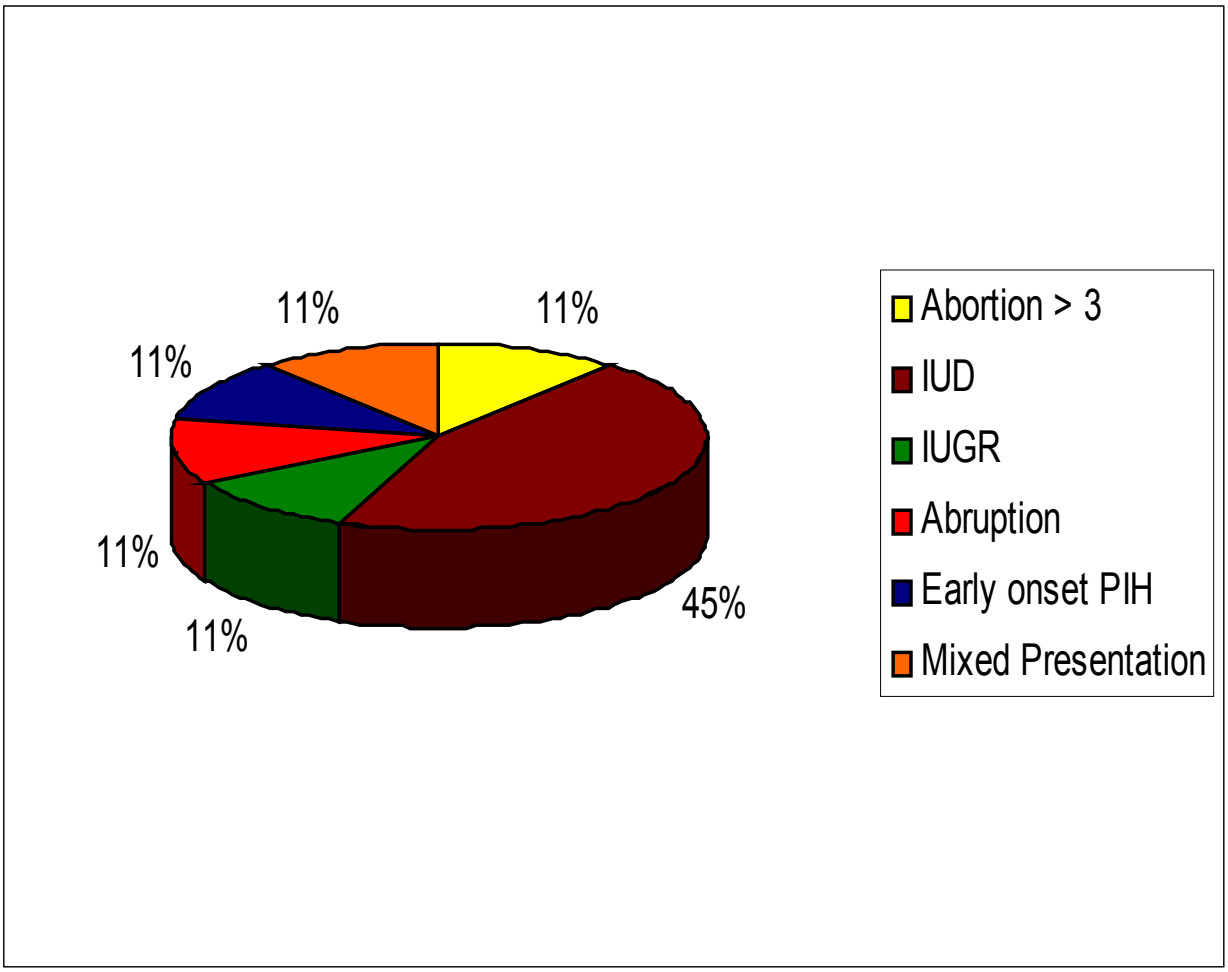



**TABLE VIII**

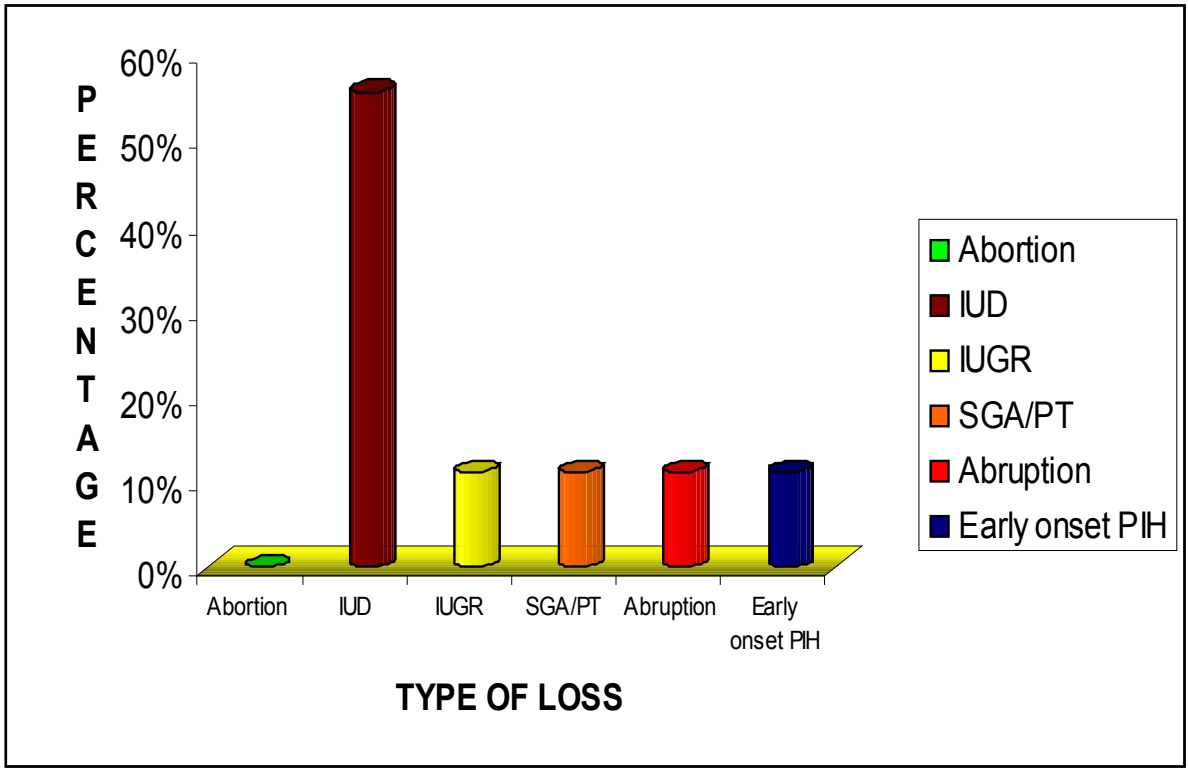
**PRESENT OBSTETRIC OUTCOME IN POSITIVE CASES**

| Type of Loss | No. of Cases | Percentage |
|---|---|---|
| Abortion | - | - |
| Intrauterine demise | 10 | 55.5% |

| | | |
|---|---|---|
| Live Births | 4 | 22.2% |
| 1. Intrauterine growth retardation | 2 | 11.1% |
| 2. SGA/PT | 2 | 11.1% |
| Abruption | 2 | 11.1% |
| Early onset PIH | 2 | 11.1% |

Live Birth was seen in 22.2% of cases, but included Small for gestational babies (SGA) babies/Intrauterine growth retardation babies and had an episode of abruption too. Intrauterine demises were seen in 55.5% of cases. 11.1% were intrauterine growth retarded, recurrent preeclampsia and Abruption. None of the cases ended up with abortions.

## PRESENT OBSTETRIC OUTCOME IN POSITIVE CASES


PERCENTAGE
60%
50%
40%
30%
20%
10%
0%
Abortion
IUD
IUGR
SGA/PT
Abruption
Early onset PIH
TYPE OF LOSS
Abortion
IUD
IUGR
SGA/PT
Abruption
Early onset PIH

# DISCUSSION

DISCUSSION

This study was undertaken to analyse the following facts;

a) The prevalence of Anti-Cardiolipin antibodies in patients with unexplained pregnancy loss.
b) The importance of screening patients with bad obstetric history for anticardiolipin antibodies.
c) Criteria for selection for screening of these patients
d) The kind of obstetric outcome that is more common in anticardiolipin antibody positive women.
e) Follow up the rest of the pregnancy to see the pregnancy outcome.

Two Hundred patients with unexplained pregnancy loss in the past were taken up for study in the Govt. RSRM lying-in hospital, attached to Stanley Medical College. These women were thoroughly screened for Diabetes, syphilis, urinary tract infections, Torch infections before subjecting them for anti cardiolipin anti body screening.

The criteria for screening women for anticardiolipin antibodies were,

i) Recurrent miscarriage ≥3

ii) Second or third trimester pregnancy loss which includes all IUD's

iii) Placental abruption

iv) H/O Early onset preeclampsia

v) Intrauterine growth retardation

vi) SLE

vii) Thrombocytopenia

None of the patients we selected had either SLE or thrombocytopenia.

The mean age of the patient in this study was 25. Maximum number of patients belonged to the age group [21-25]. The youngest patient in our study was 18yrs old and eldest 35yrs.

In DERKSON et al.'s study of anti cardiolipin antibodies in recurrent pregnancy loss, the mean age was 31 yrs. In Friedman's study mean age was 32 yrs. The increase in incidence in younger patients in this study is probably due to the early marriage in our society.

39% of patients in this study were II gravidae followed closely by III gravidae [31%]. The increase in $2^{nd}$ gravidae in our study is probably because of the criteria taken for selection. Even $2^{nd}$ (OR) $3^{rd}$ trimester loss was taken as a criterion for screening and this resulted in increase in number of $2^{nd}$ gravidae.

Almost 79% of patients screened were in their 3rd trimesters, which probably is due to our patients presenting more in term either with false or true labour pains. (i.e.) most of them were unbooked cases.

Among the 200 women screened, 18 turned out to be positive for IgG class of anticardiolipin antibodies. This 9% is quite a number compared to other studies.

RAI et al. studied women with recurrent miscarriages and showed a prevalence of 5.5% of anticardiolipin antibody positive in them.[32] LYNCH et al. in his study showed a prevalence of 15.8% of anticardiolipin antibodies in women with intrauterine fetal deaths. [36] EROGLU et al. in this study suggested that the prevalence of anticardiolipin antibody is very low in first trimester losses and is not very significant. [33] But MACLEAN and colleagues in their study proved that there was a prevalence of 8.2% in first trimester losses. [34]

But most of these studies have shown the prevalence of anticardiolipin antibodies in individual adverse outcomes either in miscarriages or in

intrauterine deaths etc. But our study shows the prevalence of anticardiolipin antibodies in a high risk population which includes recurrent abortions, intrauterine deaths, intrauterine growth retardation etc.

According to KALRA et al., anticardiolipin antibody IgM is not associated with first trimester recurrent abortion. [15] But as per LOCKWOOD et al., (1986) in his study of 55 patients showed that 18% of women with poor obstetric outcome had raised level of anticardiolipin antibody IgM. [39]

Eve Scopelitis et al., did a study in African American population and concluded that positivity for anticardiolipin antibodies is associated with an increased risk for fetal loss and not for other complications for pregnancy. [16] Velayutha Prabhu et al in his study of in their study of 155 patients with recurrent spontaneous abortions concluded that the levels of anticardiolipin antibody IgG were detected as 40%[17.]

Anne Lynch et al did a study to determine if the presence of antiphospholipid antibody in healthy pregnant women in associated with adverse pregnancy outcome including

1. Intra uterine fetal loss

2. Maternal pregnancy complications
3. Low birth weight
4. Low 5 min apgar score

The study concluded that patients with elevated APL levels at their initial prenatal visit had an increased fetal loss but no increase in maternal, pregnancy complications, low birth weight or low apgar score. Immunoglobulin G anticardiolipin antibody was the only single test of APL significantly associated with fetal loss. [18]

Mary Bird Sall et al., in her study assessed the relationship between antiphospholipid antibodies and recurrent miscarriage, fetal death, and pregnancy complications-placental abruption, fetal growth retardation and preeclampsia. The subjects were 81 women with a history of 3 or more miscarriages, 62 with a history of fetal death in the index pregnancy, 105 with a poor obstetric history or pregnancy complications and 13 with systemic lupus erythematosus.

Antiphospholipid antibodies were found in 41% of women with history of recurrent miscarriage, 29% with the history of recent fetal death, 19% with a poor obstetric history and she concluded that patients with the

above pregnancy disorders should be tested for antiphospholipid antibodies because of the risk conferred on their fetus by their presence and to expand the treatment options. [22]

Kaneria et al., did a pilot study in 50 patients of bad obstetric history for the presence of lupus anticoagulant and anticardiolipin antibodies. LA alone was positive in 6 patients (12%) and ACLA alone was positive in 14 patients (28%) while both LA and ACLA were positive in 3 patients (6%)[20]

Kumar et al., did a study with 150 patients with recurrent pregnancy loss in South India and found 11 patients positive for ACLA and suggested the usefulness of screening for these antibodies as a mandatory routine for instituting efficient therapeutic regimes for a successful outcome of pregnancy. [2]

The commonest type of obstetric history in our antiphospholipid antibody positive women is intrauterine death which occupies a 55.5% followed by Intrauterine growth retardation, abruption and early onset preeclampsia all of which are around 11.1%.

This probably tallies well with BOCCIOLONE et al's observation that intrauterine late foetal death is more common in anti cardiolipin antibody positive than in lupus anti coagulant positive. Recurrent spontaneous abortions according to him were more common in lupus anticoagulant positive than anticardiolipin antibodies positive. In this observation 11% of anticardiolipin antibody positive had intrauterine demises.

DERIK'S et at., in his retrospective analysis found out that 80% of women with anticardiolipin antibodies had at least one fetal death compared with less than 25% of women without. The specificity according to him for the presence of anti phospholipid antibodies in patients with recurrent pregnancy loss was 76%. In contrast, 2 or earlier first trimester losses had a specificity of only 6%. Thus they concluded that foetal death is more characteristic of the type of loss experienced by patients with recurrent pregnancy loss than early first trimester pregnancy loss in patient with Anticardiolipin antibody[37]. Live birth rate in this study was around 22.2% (i.e. 4 out of 18 patients) where 2 were SGA and preterm and 1 was an intrauterine growth retarded baby.

# SUMMARY

## SUMMARY

Two Hundred pregnant women with previous unexplained pregnancy loss were taken for this study, to detect for the presence of anticardiolipin antibodies, with the idea that these antibodies might be the one responsible

for their pregnancy loss. This suggestion is made after screening them for all other possible causes.

1). In the study group eighteen of them tested positive for anticardiolipin antibodies. In this positive group, more than half of them (55.5%) were 2nd gravidae.

2). The Mean age of the patient in this study was 25.

3). Maximum number of patients belongs to the age group of 21-25 years.

4). The youngest patient in our study was 18 years old and eldest 35 years.

5). 39% of patients in this study were second Gravida

6). A majority of them [55.5%] presented to us only in the third trimester either with c/o loss of foetal movements, bleeding p/v or just with onset of labour contractions.

7). An analysis of the previous obstetric history revealed that intrauterine deaths were the predominant presentation [45%] followed next only by other presentations like abortions, abruptions etc.

8). A follow up of the present pregnancy also showed that IUD's were in the forefront (55.5%)

9). Live birth rate was 22.2% which included 11.1% intrauterine growth retardation, 11.1% small for gestational age / preterm.

10). 11.1% had abruption, 11.1% had early onset PIH.

# CONCLUSION

CONCLUSION

In this study, women who are positive for anticardiolipin antibody have a significant risk of reproductive failure and adverse pregnancy

outcome. The incidence of recurrent abortion, foetal death and intrauterine growth retardation is significant.

THIS STUDY PROVES THAT ANTICARDIOLIPIN ANTIBODIES ARE ASSOCIATED WITH SIGNIFICANT FOETAL LOSS. SECOND AND THIRD TRIMESTER LOSSES IN THE FORM OF INTRAUTERINE DEATHS WERE THE COMMONEST OBSTETRIC OUTCOME.

Women with unexplained loss should essentially be screened for anticardiolipin antibodies and this should be a part of investigation protocol followed. As a best management practice, screening should be done earlier, as soon as the pregnancy test becomes positive.

The best approach, to be professed is to form multidisciplinary teams for management methods to exclude other causes of pregnancy loss, lay down the proper criterion for screening for ACL, and select the perfect regimen which will cause the least harm to both the mother and the fetus.

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# PROFORMA

NAME: AGE: IPNO: UNIT:

ADDRESS: OCCUPTION:

INCOME: SOCIOECONOMIC STATUS:

DOA:

PARITY INDEX:

LMP EDD PERIOD OF GESTN

PRESENTING COMPLAINTS:

MENSTURAL HISTORY:

MARITAL HISTORY:

OBSTETRIC HISTROY:

PREVIOUS OBSTETRIC HISTORY:

ABORTION::

NUMBER

GESTATIONAL AGE

CONGENITAL ANOMALY

TYPE

STILL BORN::

NUMBER

GESTATIONAL AGE

FRESH OR MACERATED

SEX

WEIGHT

CONGENITAL ANOMALY

NEONATAL DEATH::

NUMBER

CAUSE

PRETERM BIRTH::

NUMBER

GESTATIONAL AGE

H/O PROM

OUTCOME

LIVE BIRTH::

- NUMBER
- WEIGHT
- SEX
- CONGENITAL ANOMALY
- PRESENT HEALTH

HISTORY OF ANY PREDISPOSING FACTOR TO BOH

HISTORY OF PRESENT PREGNANCY

PAST HISTORY

PERSONAL HISTORY

FAMILY HISTORY

GENERAL EXAMINATION

| | |
|---|---|
| HEIGHT | WEIGHT |
| PALLOR | ODEMA |
| PULSE | BP |
| BREAST | THYROID |
| CVS | CNS |

OBSTERTIC EXAMINATION

INVESTIGATION:

URINE ROUTINE EXAMINATION

COMPLETE HEMOGRAM

BLOOD GROUPING & Rh TYPING:

VDRL:

BLOOD SUGAR/GTT::

BLOOD UREA::

S.CREATININE::

SERUM URIC ACID::

S.BILIRUBIN::

BT ::

CT::

ULTRASOUND

ELISA FOR IgG ACA

PREGNANCY OUTCOME

# MASTERCHART

| Sl.No | Name | Age | Ipno | Unit | Parity | GA/Week | ACLA | Pregnancy Outcome |
|---|---|---|---|---|---|---|---|---|
| 1 | Sirajunisha | 23 | 34449 | II | $G_4P_2L_0A_1$ | 8w | **Positive** | SGA |
| 2 | Kondammal | 28 | 10215 | I | $G_5P_4L_1$ | 12w | Negative | Abortion |
| 3 | Devi | 26 | 29975 | II | $G_2P_1L_0$ | 24w | Negative | Live Baby |
| 4 | Geetha | 22 | 18827 | IV | $G_3P_1L_0A_1$ | 36w | Negative | IUGR |
| 5 | Bhuvaneswari | 23 | 17190 | IV | $G_2P_1L_0$ | 34w | Negative | IUGR |
| 6 | Aruna | 20 | 7646 | III | $G_2P_1L_0$ | 34w | Negative | IUD |
| 7 | Ammu | 23 | 26430 | I | $G_3P_2L_0$ | 28w | Negative | Live Baby |
| 8 | Karishma | 28 | 22656 | IV | $G_3P_2L_0$ | 32w | Negative | PIH |
| 9 | Kanjana | 25 | 34261 | IV | $G_2P_1L_0$ | 36w | Negative | Live Baby |
| 10 | Shakila Devi | 19 | 26025 | I | $G_3P_2L_0$ | 18w | Negative | Live Baby |
| 11 | Madhavi | 23 | 26200 | II | $G_2P_1L_0$ | 22w | **Positive** | Abruption |
| 12 | Gomathi | 22 | 22894 | IV | $G_3P_1L_0A_1$ | 34w | Negative | Live Baby |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 13 | Sulochana | 20 | 300591 | I | $G_6P_3L_1A_2$ | 32w | Negative | IUGR |
| | | | | | | | | |
| 14 | Shakila | 29 | 28376 | II | $G_5A_4$ | 34w | Negative | Live Baby |
| | | | | | | | | |
| 15 | Multhani | 22 | 36074 | IV | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 16 | Yamuna | 29 | 36050 | IV | $G_2P_1L_0$ | 36w | Negative | Live Baby |
| | | | | | | | | |
| 17 | Saraswathy | 26 | 984 | V | $G_2P_1L_0$ | 34w | Negative | Live Baby |
| | | | | | | | | |
| 18 | Tamilselvi | 23 | 14445 | III | $G_5P_2L_1A_2$ | 30w | Negative | IUGR |
| | | | | | | | | |
| 19 | Yasodha | 30 | 37021 | I | $G_2P_1L_0$ | 20w | **Positive** | IUD |
| | | | | | | | | |
| 20 | Renuka | 22 | 38723 | II | $G_3P_1L_0A_1$ | 28w | Negative | SGA |
| | | | | | | | | |
| 21 | Parvathy | 32 | 19559 | IV | $G_2P_1L_0$ | 29w | Negative | Live Baby |
| 22 | Selvi | 24 | 17321 | IV | $G_2P_1L_0$ | 11w | Negative | Live Baby |
| | | | | | | | | |
| 23 | Saroja | 23 | 19645 | IV | $G_4P_1L_1A_2$ | 33w | Negative | Live Baby |
| | | | | | | | | |
| 24 | Eswari | 22 | 7143 | V | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 25 | Varalakshmi | 32 | 17390 | I | $G_4P_2L_1A_1$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 26 | Sumathi | 33 | 20897 | II | $G_4A_3$ | 34w | Negative | Early PIH |
| | | | | | | | | |
| 27 | Padmavathi | 25 | 18675 | IV | PRIMI | 22w | Negative | Live Baby |
| | | | | | | | | |
| 28 | Savithri | 18 | 16421 | I | $G_2P_1L_0$ | 34w | Negative | Abruption |
| | | | | | | | | |
| 29 | Kupamma | 20 | 7644 | II | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 30 | Ammu | 22 | 8612 | III | $G_4P_2L_1A_1$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 31 | Aruna | 24 | 11242 | I | $G_2P_1L_0$ | 36w | Negative | Live baby |
| | | | | | | | | |
| 32 | Jency | 26 | 11264 | IV | $G_2P_1L_0$ | 28w | **Positive** | IUD |
| | | | | | | | | |
| 33 | Wahidh Begum | 28 | 16241 | V | $G_3P_1L_0A_1$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 34 | Kanniyamal | 29 | 18926 | II | $G_2P_1L_0$ | 31w | Negative | Live Baby |
| | | | | | | | | |
| 35 | Mari | 28 | 19321 | III | $G_3P_1L_0A_1$ | 32w | Negative | Live Baby |
| | | | | | | | | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 36 | Muthamal | 30 | 6499 | IV | $G_2P_1L_0$ | 32w | Negative | IUGR |
| 37 | Jeyanthi | 31 | 73641 | I | $G_4P_2L_0A_1$ | 11w | Negative | Live Baby |
| 38 | Mani | 20 | 11211 | II | $G_3P_2L_0$ | 35w | Negative | Live Baby |
| 39 | Banu | 22 | 20629 | I | PRIMI | 34w | Negative | Live Baby |
| 40 | Raayamma | 20 | 20612 | I | $G_2P_1L_0$ | 33w | Negative | Live Baby |
| 41 | Kavitha | 26 | 15941 | IV | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| 42 | Rekha | 24 | 16921 | III | $G_3P_2L_0$ | 33w | Negative | SGA |
| 43 | Priya | 18 | 10432 | II | $G_3P_1L_0A_1$ | 31w | Negative | Live Baby |
| 44 | Sivagami | 19 | 20672 | III | $G_2P_1L_1$ | 30w | **Positive** | IUD |
| 45 | Usha | 21 | 11324 | III | $G_2P_1L_0$ | 11w | Negative | Live Baby |
| 46 | Devi | 23 | 11431 | I | $G_2P_1L_0$ | 35w | Negative | Live Baby |
| 47 | Shankari | 24 | 16789 | I | $G_2P_1L_0$ | 29w | Negative | Live Baby |
| 48 | Krishna | 32 | 9994 | I | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| 49 | Yuvarani | 22 | 9443 | III | $G_2P_1L_0$ | 31w | Negative | Live Baby |
| 50 | Sandhya | 23 | 17621 | III | PRIMI | 32w | Negative | Live Baby |
| 51 | Ramya | 26 | 42611 | I | $G_3P_2L_1$ | 34w | Negative | IUGR |
| 52 | Rahini | 27 | 9734 | II | $G_3P_2L_0$ | 32w | Negative | SGA |
| 53 | Katammal | 28 | 162112 | V | $G_2P_1L_0$ | 30w | Negative | Live Baby |
| 54 | Laskhmi | 22 | 10621 | IV | $G_2P_1L_0$ | 32w | **Positive** | IUGR |
| 55 | Seetha | 26 | 13622 | III | $G_3P_1L_0A_1$ | 30w | Negative | Live Baby |
| 56 | Bavani | 24 | 11341 | I | $G_2P_1L_0$ | 34w | Negative | IUGR |
| 57 | Dhanalaskhmi | 25 | 1246 | I | $G_2P_1L_0$ | 29w | Negative | IUD |
| 58 | Bhuvana | 18 | 10642 | III | PRIMI | 30w | Negative | SGA |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 59 | Chitra | 19 | 96212 | I | $G_4P_1L_1A_2$ | 11w | Negative | Live Baby |
| | | | | | | | | |
| 60 | SivaSankari | 21 | 94211 | II | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 61 | Kumari | 29 | 9334 | III | $G_2P_1L_0$ | 33w | Negative | Abruption |
| | | | | | | | | |
| 62 | Aaandal | 31 | 10421 | III | $G_2P_1L_0$ | 7w | Negative | Live Baby |
| | | | | | | | | |
| 63 | Priyadarshini | 30 | 16231 | I | $G_3P_2L_0$ | 34w | Negative | Live Baby |
| | | | | | | | | |
| 64 | Shakunthala | 24 | 1033 | I | $G_4P_3L_1$ | 34w | **Positive** | Early Onset PIH |
| | | | | | | | | |
| 65 | Amala | 30 | 6080 | III | $G_3P_1L_0A_1$ | 32w | Negative | Abortion |
| | | | | | | | | |
| 66 | Meenatchi | 23 | 18779 | II | $G_4A_3$ | 34w | Negative | Live Baby |
| | | | | | | | | |
| 67 | Rathi | 27 | 15995 | II | $G_4P_1L_0A_2$ | 30w | **Positive** | IUD |
| | | | | | | | | |
| 68 | Saraswati | 21 | 12800 | I | $G_5P_2L_1A_2$ | 28w | Negative | Live Baby |
| | | | | | | | | |
| 69 | Ameena | 24 | 17658 | I | $G_5P_2L_1A_2$ | 32w | Negative | SGA |
| 70 | Komala | 22 | 6460 | III | $G_3P_2L_0$ | 32w | Negative | IUGR |
| | | | | | | | | |
| 71 | Zareena | 25 | 20577 | IV | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 72 | Shajidha | 23 | 20558 | IV | $G_3P_1L_0A_1$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 73 | Poongodi | 23 | 6479 | III | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 74 | Jayanthi | 29 | 12645 | I | $G_3P_1L_0A_1$ | 32w | Negative | IUGR |
| | | | | | | | | |
| 75 | Datchayini | 30 | 24890 | III | $G_4P_1L_1A_2$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 76 | Maleshwari | 22 | 15014 | IV | $G4P_2L_1A_1$ | 34w | Negative | IUGR |
| | | | | | | | | |
| 77 | Manju | 20 | 33385 | I | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| | | | | | | | | |
| 78 | Komala | 31 | 19041 | II | $G_3P_1L_0A_1$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 79 | Ameena | 24 | 17658 | IV | $G_5P_2L_1A_2$ | 34w | Negative | IUD |
| | | | | | | | | |
| 80 | Latha | 26 | 6513 | I | $G4P_2L_1A_1$ | 34w | Negative | Live Baby |
| | | | | | | | | |
| 81 | Amaravathi | 20 | 18063 | III | $G_6A_5$ | 30w | **Positive** | IUD |
| | | | | | | | | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 82 | Anandhi | 23 | 7810 | I | $G_4P_2L_0A_1$ | 34w | Negative | Live Baby |
| 83 | Masthani | 22 | 10090 | IV | $G_4P_1L_1A_2$ | 32w | Negative | Live Baby |
| 84 | Usha | 20 | 9946 | I | $G_4A_3$ | 30w | Negative | SGA |
| 85 | Manjula | 35 | 9993 | II | $G_3P_1L_0A_1$ | 34w | Negative | Live Baby |
| 86 | Lashmikantha | 26 | 12746 | I | $G_2P_1L_0$ | 8w | Negative | Live Baby |
| 87 | Saranya | 28 | 14721 | IV | $G_4P_1L_0A_2$ | 32w | Negative | Live Baby |
| 88 | Sajitha Banu | 27 | 14521 | I | $G_2P_1L_0$ | 28w | Negative | Live Baby |
| 89 | Chandrakala | 25 | 12726 | IV | $G_2P_1L_0$ | 34w | Negative | Preterm |
| 90 | Angammal | 24 | 83851 | V | $G_3P_1L_0A_1$ | 34w | Negative | IUGR |
| 91 | Beulah | 25 | 10080 | III | $G_3P_1L_0A_1$ | 38w | Negative | Live Baby |
| 92 | Murugammal | 23 | 45141 | II | $G_2P_1L_0$ | 36w | Negative | Live Baby |
| 93 | Shyamala | 24 | 18841 | III | $G_3P_2L_0$ | 34w | Negative | Live Baby |
| 94 | Kalavathy | 25 | 26212 | IV | $G_3P_1L_0A_1$ | 34w | Negative | Live Baby |
| 95 | Nirmala | 24 | 34242 | II | $G_3P_1L_0A_1$ | 32w | Negative | IUD |
| 96 | Rajeswari | 23 | 42612 | III | $G_2P_1L_0$ | 28w | Negative | Live Baby |
| 97 | Rekha | 24 | 14321 | I | $G_2P_1L_0$ | 32w | Negative | Abortion |
| 98 | Amudha | 25 | 33862 | I | $G_3P_1L_0A_1$ | 34w | Negative | SGA |
| 99 | Vatchala | 24 | 15262 | III | $G_4A_3$ | 32w | Negative | Live Baby |
| 100 | Bharathi | 23 | 16241 | IV | $G_2P_1L_0$ | 12w | Negative | Live Baby |
| 101 | Ambiga | 23 | 14449 | II | $G_4P_2L_0A_1$ | 8w | **Positive** | SGA |
| 102 | Pushpanjali | 28 | 16777 | I | $G_5P_4L_1$ | 12w | Negative | Abortion |
| 103 | Manju | 26 | 19876 | II | $G_2P_1L_0$ | 24w | Negative | Live Baby |
| 104 | Nasiya | 22 | 13327 | IV | $G_3P_1L_0A_1$ | 36w | Negative | IUGR |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 105 | Rajalakshmi | 23 | 17233 | IV | $G_2P_1L_0$ | 34w | Negative | IUGR |
| 106 | Gracy Shoba | 20 | 17646 | III | $G_2P_1L_0$ | 34w | Negative | IUD |
| 107 | Sumithra | 23 | 26430 | I | $G_3P_2L_0$ | 28w | Negative | Live Baby |
| 108 | Gunavathy | 28 | 22656 | IV | $G_3P_2L_0$ | 32w | Negative | PIH |
| 109 | Selvi | 25 | 34261 | IV | $G_2P_1L_0$ | 36w | Negative | Live Baby |
| 110 | Rajeswari | 19 | 26025 | I | $G_3P_2L_0$ | 18w | Negative | Live Baby |
| 111 | Farziya Banu | 23 | 26200 | II | $G_2P_1L_0$ | 22w | **Positive** | Abruption |
| 112 | Amulu | 22 | 22894 | IV | $G_3P_1L_0A_1$ | 34w | Negative | Live Baby |
| 113 | Lalitha | 20 | 300591 | I | $G_6P_3L_1A_2$ | 32w | Negative | IUGR |
| 114 | Kasthuri | 29 | 28376 | II | $G_5A_4$ | 34w | Negative | Live Baby |
| 115 | Ellammal | 22 | 36074 | IV | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| 116 | Rani | 29 | 36050 | IV | $G_2P_1L_0$ | 36w | Negative | Live Baby |
| 117 | Sulochana | 26 | 984 | V | $G_2P_1L_0$ | 34w | Negative | Live Baby |
| 118 | Sarala | 23 | 14445 | III | $G_5P_2L_1A_2$ | 30w | Negative | IUGR |
| 119 | Nagamuthu | 30 | 37021 | I | $G_2P_1L_0$ | 20w | **Positive** | IUD |
| 120 | Usha | 22 | 38723 | II | $G_3P_1L_0A_1$ | 28w | Negative | SGA |
| 121 | Kumari | 32 | 19559 | IV | $G_2P_1L_0$ | 29w | Negative | Live Baby |
| 122 | Kamala | 24 | 17321 | IV | $G_2P_1L_0$ | 11w | Negative | Live Baby |
| 123 | Valarmathi | 23 | 19645 | IV | $G_4P_1L_1A_2$ | 33w | Negative | Live Baby |
| 124 | Kalyani | 22 | 7143 | V | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| 125 | Sabitha | 32 | 17390 | I | $G_4P_2L_1A_1$ | 30w | Negative | Live Baby |
| 126 | Anjali devi | 33 | 20897 | II | $G_4A_3$ | 34w | Negative | Early PIH |
| 127 | Sudha | 25 | 18675 | IV | PRIMI | 22w | Negative | Live Baby |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 128 | Srideepa | 18 | 16421 | I | $G_2P_1L_0$ | 34w | Negative | Abruption |
| 129 | Parameswari | 20 | 7644 | II | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| 130 | Muniammal | 22 | 8612 | III | $G_4P_2L_1A_1$ | 30w | Negative | Live Baby |
| 131 | Kumudha | 24 | 11242 | I | $G_2P_1L_0$ | 36w | Negative | Live baby |
| 132 | Indiramathy | 26 | 11264 | IV | $G_2P_1L_0$ | 28w | **Positive** | IUD |
| 133 | Thareswari | 28 | 16241 | V | $G_3P_1L_0A_1$ | 30w | Negative | Live Baby |
| 134 | Vijaya | 29 | 18926 | II | $G_2P_1L_0$ | 31w | Negative | Live Baby |
| 135 | Radhika | 28 | 19321 | III | $G_3P_1L_0A_1$ | 32w | Negative | Live Baby |
| 136 | Gauthami | 30 | 6499 | IV | $G_2P_1L_0$ | 32w | Negative | IUGR |
| 137 | Sargu | 31 | 73641 | I | $G_4P_2L_0A_1$ | 11w | Negative | Live Baby |
| 138 | Anaswathy | 20 | 11211 | II | $G_3P_2L_0$ | 35w | Negative | Live Baby |
| 139 | Padma | 22 | 20629 | I | PRIMI | 34w | Negative | Live Baby |
| 140 | Eswari | 20 | 20612 | I | $G_2P_1L_0$ | 33w | Negative | Live Baby |
| 141 | Tamilarasi | 26 | 15941 | IV | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| 142 | Soundari | 24 | 16921 | III | $G_3P_2L_0$ | 33w | Negative | SGA |
| 143 | Vaali | 18 | 10432 | II | $G_3P_1L_0A_1$ | 31w | Negative | Live Baby |
| 144 | Jayanthi | 19 | 20672 | III | $G_2P_1L_1$ | 30w | **Positive** | IUD |
| 145 | Saradha | 21 | 11324 | III | $G_2P_1L_0$ | 11w | Negative | Live Baby |
| 146 | Yasodha | 23 | 11431 | I | $G_2P_1L_0$ | 35w | Negative | Live Baby |
| 147 | Jayammal | 24 | 16789 | I | $G_2P_1L_0$ | 29w | Negative | Live Baby |
| 148 | Gowri | 32 | 9994 | I | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| 149 | Saraswathi | 22 | 9443 | III | $G_2P_1L_0$ | 31w | Negative | Live Baby |
| 150 | Latha | 23 | 17621 | III | PRIMI | 32w | Negative | Live Baby |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 151 | Poongodi | 26 | 42611 | I | $G_3P_2L_1$ | 34w | Negative | IUGR |
| 152 | Chandra | 27 | 9734 | II | $G_3P_2L_0$ | 32w | Negative | SGA |
| 153 | Saroja | 28 | 162112 | V | $G_2P_1L_0$ | 30w | Negative | Live Baby |
| 154 | Tamilselvi | 22 | 15461 | IV | $G_2P_1L_0$ | 32w | **Positive** | IUGR |
| 155 | Seetha | 26 | 13622 | III | $G_3P_1L_0A_1$ | 30w | Negative | Live Baby |
| 156 | Sathya | 24 | 11341 | I | $G_2P_1L_0$ | 34w | Negative | IUGR |
| 157 | Gayathri | 25 | 1246 | I | $G_2P_1L_0$ | 29w | Negative | IUD |
| 158 | Muthamma | 18 | 10642 | III | PRIMI | 30w | Negative | SGA |
| 159 | Manjula | 19 | 96212 | I | $G_4P_1L_1A_2$ | 11w | Negative | Live Baby |
| 160 | Sathiya | 21 | 94211 | II | $G_2P_1L_0$ | 32w | Negative | Live Baby |
| 161 | Kumari | 29 | 9334 | III | $G_2P_1L_0$ | 33w | Negative | Abruption |
| 162 | Vijaya | 31 | 10421 | III | $G_2P_1L_0$ | 7w | Negative | Live Baby |
| 163 | Priyadarshini | 30 | 16231 | I | $G_3P_2L_0$ | 34w | Negative | Live Baby |
| 164 | Sheeba | 24 | 12673 | I | $G_4P_3L_1$ | 34w | **Positive** | Early Onset PIH |
| 165 | Nithya | 30 | 6080 | III | $G_3P_1L_0A_1$ | 32w | Negative | Abortion |
| 166 | Ramadevi | 23 | 18779 | II | $G_4A_3$ | 34w | Negative | Live Baby |
| 167 | Sahana | 27 | 15454 | II | $G_4P_1L_0A_2$ | 30w | **Positive** | IUD |
| 168 | Sree | 21 | 12800 | I | $G_5P_2L_1A_2$ | 28w | Negative | Live Baby |
| 169 | Rani | 24 | 17658 | I | $G_5P_2L_1A_2$ | 32w | Negative | SGA |
| 170 | Komala | 22 | 6460 | III | $G_3P_2L_0$ | 32w | Negative | IUGR |
| 171 | Zareena | 25 | 20577 | IV | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| 172 | Shajidha | 23 | 20558 | IV | $G_3P_1L_0A_1$ | 30w | Negative | Live Baby |
| 173 | Poongodi | 23 | 6479 | III | $G_3P_2L_0$ | 30w | Negative | Live Baby |

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|---|---|---|---|---|---|---|---|---|
| 174 | Jayanthi | 29 | 12645 | I | $G_3P_1L_0A_1$ | 32w | Negative | IUGR |
| 175 | Datchayini | 30 | 24890 | III | $G_4P_1L_1A_2$ | 32w | Negative | Live Baby |
| 176 | Maleshwari | 22 | 15014 | IV | $G4P_2L_1A_1$ | 34w | Negative | IUGR |
| 177 | Manju | 20 | 33385 | I | $G_3P_2L_0$ | 30w | Negative | Live Baby |
| 178 | Komala | 31 | 19041 | II | $G_3P_1L_0A_1$ | 32w | Negative | Live Baby |
| 179 | Ameena | 24 | 17658 | IV | $G_5P_2L_1A_2$ | 34w | Negative | IUD |
| 180 | Latha | 26 | 6513 | I | $G4P_2L_1A_1$ | 34w | Negative | Live Baby |
| 181 | Sarasu | 20 | 19600 | III | $G_6A_5$ | 30w | **Positive** | IUD |
| 182 | Anandhi | 23 | 7810 | I | $G_4P_2L_0A_1$ | 34w | Negative | Live Baby |
| 183 | Masthani | 22 | 10090 | IV | $G_4P_1L_1A_2$ | 32w | Negative | Live Baby |
| 184 | Usha | 20 | 9946 | I | $G_4A_3$ | 30w | Negative | SGA |
| 185 | Manjula | 35 | 9993 | II | $G_3P_1L_0A_1$ | 34w | Negative | Live Baby |
| 186 | Lashmikantha | 26 | 12746 | I | $G_2P_1L_0$ | 8w | Negative | Live Baby |
| 187 | Saranya | 28 | 14721 | IV | $G_4P_1L_0A_2$ | 32w | Negative | Live Baby |
| 188 | Sajitha Banu | 27 | 14521 | I | $G_2P_1L_0$ | 28w | Negative | Live Baby |
| 189 | Chandrakala | 25 | 12726 | IV | $G_2P_1L_0$ | 34w | Negative | Preterm |
| 190 | Kumudha | 24 | 83851 | V | $G_3P_1L_0A_1$ | 34w | Negative | IUGR |
| 191 | Rajeswari | 25 | 10080 | III | $G_3P_1L_0A_1$ | 38w | Negative | Live Baby |
| 192 | Durgadevi | 23 | 45141 | II | $G_2P_1L_0$ | 36w | Negative | Live Baby |
| 193 | Sangeetha | 24 | 18841 | III | $G_3P_2L_0$ | 34w | Negative | Live Baby |
| 194 | Jeevitha | 25 | 26212 | IV | $G_3P_1L_0A_1$ | 34w | Negative | Live Baby |
| 195 | Vasanthi | 24 | 34242 | II | $G_3P_1L_0A_1$ | 32w | Negative | IUD |
| 196 | Anandhi | 23 | 42612 | III | $G_2P_1L_0$ | 28w | Negative | Live Baby |

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|---|---|---|---|---|---|---|---|---|
| 197 | Sujatha | 24 | 14321 | I | $G_2P_1L_0$ | 32w | Negative | Abortion |
| | | | | | | | | |
| 198 | Priya | 25 | 33862 | I | $G_3P_1L_0A_1$ | 34w | Negative | SGA |
| | | | | | | | | |
| 199 | Bhoopathy | 24 | 15262 | III | $G_4A_3$ | 32w | Negative | Live Baby |
| | | | | | | | | |
| 200 | Kushboo | 23 | 16241 | IV | $G_2P_1L_0$ | 12w | Negative | Live Baby |